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THE RELATION OF SINUSITIS TO NEPHROSIS IN CHILDREN

A STUDY BASED ON INVESTIGATIONS AT THE CHILDREN'S HOSPITAL, LOS ANGELES, 1929 *

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The pathologic process in true nephrosis is confined to the tubular portion of the kidney. All grades of tubular changes may be noted. The cells become granular in an early stage, and finally shed off and regenerate in a lower type as the disease progresses.

A pure tubular nephrosis is rare, however, as secondary glomerular changes and interstitial changes are frequent. The glomerular capsule becomes thickened; later the glomerular tuft becomes adherent to the capsule, and finally there is entire obliteration of the tuft. Interstitial changes with round cell infiltration also take place. The condition is then one of chronic parenchymatous nephritis or a mixed nephritis and nephrosis.

A somewhat similar picture is sometimes obtained when, following acute glomerular nephritis, as sometimes results from scarlet fever, a secondary degeneration of the tubules occurs. The etiologic agent in this case may be bacterial, in which case the changes in the kidney are decidedly focal, or it may be a toxin circulating in the blood, the changes then being quite diffuse.

The etiologic factor in true nephrosis is generally believed to be a toxin arising from a focus of infection.

Clinically, nephrosis is characterized by edema, low urinary output, albuminuria, hyaline and granular casts in the urine, nonretention of nonprotein nitrogen, normal blood pressure and normal phthalein output. There are few, if any, red blood cells or pus cells in the urine.

Marriott [1] stated that one of the most important and clearcut manifestations of infection in the nose and throat of children is tubular and glomerular nephritis. He also stated that nephritis is invariably associated with infection, and most frequently with infection of the nose and throat. In practically all cases of nephrosis in children under his observation, he found infection of the nasal sinuses, particularly the

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1. Marriott, McKim: Ann. Otol., Rhin. & Laryng. **36**:686, 1927.

antrums. *Staphylococcus hemolyticus* was found to be the most common infecting organism. He reported remarkable effects with treatment, one patient losing 15 pounds (6.8 Kg.) of fluid following the drainage of an infected sinus. He has followed several cases for a number of years with no remaining evidence of nephrosis. In some cases, however, treatment brought about only temporary and symptomatic improvement, because it was not always possible to eradicate the infection completely. He expressed the belief that infections of long standing are likely to lead to permanent changes in the kidney, and that the most to be expected is the prevention of further damage and progressive changes.

Clausen [2] emphasized the importance of staphylococcus infections as etiologic factors in nephrosis. In one of his cases, staphylococcus empyema, in another, staphylococcus myelitis, and in a third, an infected burn, preceded the renal symptoms. Since drainage of the infected locality was followed by prompt cessation of symptoms, he concluded that toxic substances were absorbed from the focus.

Clausen also called attention to the susceptibility of these patients to infection. The chief causes of death have been peritonitis, pneumonia and empyema.

In a later paper,[3] he reported results which led him to believe that infection of the nasal sinuses plays an important etiologic rôle in nephrosis. The cases reported showed definite evidence that such infection was present invariably in each of a series of unselected cases; that exacerbations of the infection were accompanied by exaggeration of the symptoms, and that successful treatment of the infection was followed by improvement or cure. Clausen expressed the belief that one of the most important consequences of nephrosis is a decreased resistance to infection of all sorts, and that this susceptibility to infection frequently leads to a sinusitis, which, in turn, aggravates the nephrosis, thus creating a vicious circle.

The diagnosis of sinusitis in children in some cases presents some difficulty. A careful study, history and observation are necessary before this diagnosis can be definitely affirmed or denied. As Marriott stated, pain, headache and purulent nasal discharge are not necessary for diagnosis, nor can roentgenograms be fully relied on, especially in infants. Marriott described the young patient, suffering from sinusitis, as one frequently pictured in tuberculosis—pale and languid, with slight afternoon temperature, and a chronic cough. Roentgenograms of the chest often show an enlarged hilus shadow and peribronchial thickening. Chronic bronchitis is often a part of the picture. Often, attacks of

2. Clausen, S. W.: Parenchymatous Nephritis: I. As a General Systemic Disorder, Am. J. Dis. Child. **29**:581 (May) 1925.

3. Clausen, S. W.: Parenchymatous Nephritis: II. Infection of the Paranasal Sinuses as Etiology, Am. J. Dis. Child. **29**:587 (May) 1925.

vomiting, occurring periodically and diagnosed as cyclic vomiting, are present. Heart murmurs and cardiac dilatation have been observed to clear up after treatment of the sinus disease. One fairly constant physical sign, according to Marriott, is enlargement of the posterior cervical group of glands, these glands only enlarging from sinus disease, ear infections and scalp infection, the last two conditions being readily eliminated. Finally, the irrigation of the antrums and the cytologic examination, as well as the culturing of the irrigating fluid, offer conclusive evidence, if positive, of sinus disease (Sewall).[4]

The treatment of the diseased sinus may be either conservative or radical. In all cases, diseased tonsils and adenoids, if present, must be removed. Eighty per cent have been cured by this means alone.[5] Because of the age of the patient, long chronicity is excluded, and therefore extensive operations are rarely necessary. Any nasal obstruction should be remedied; a warm, dry climate is recommended; autogenous vaccines have been of benefit in some cases; nasal shrinkage and irrigations, irrigations of the sinus, and in more resistant cases, window operation beneath the inferior turbinate have been reported useful. Shea[6] obtained good results by resecting a window beneath the inferior turbinate and introducing a rubber catheter. Ten per cent solution of mild silver protein was introduced three times a day for five days. If the discharge was profuse, suction was applied. The tube was removed on the fifth day, and subsequent irrigations were made with a straight needle or curved trocar. The irrigations are continued until the discharge is stopped. With a return of symptoms, irrigations are commenced again.

We have obtained good results with a Lichwitz[7] needle or a Yankauer trocar, first applying a little of 10 per cent solution of cocaine, preceded by epinephrine, beneath the inferior turbinate or in the middle meatus, as the case may be. No untoward accidents have occurred from the use of the cocaine; the small patients submit readily after their confidence is gained and prefer it to gas anesthesia. They seldom exhibit any fright after the first one or two operations. Irrigations can be performed by this method as often as desired. Irrigations have been done on patients 3 years of age with no difficulty. With a sterile needle, rubber connection, 10 cc. Luer syringes, fluid may be withdrawn for culture, or a sterile saline solution introduced and withdrawn for

4. Sewall, E. C.: Cytologic Examination of the Antrum, Arch. Otolaryng. **10**:1 (July) 1929.

5. Dean, L. W.: Ann. Otol., Rhin. & Laryng. **29**:675, 1920. Dean, L. W., and Armstrong, M.: Ibid. **29**:602, 1920.

6. Shea, J. J.: Tr. Am. Laryng. Soc., 1925, p. 186.

7. Skillern: Accessory Sinuses of the Nose, Philadelphia, J. B. Lippincott & Company, 1923, p. 168.

culture and cytologic examination. Contamination is unlikely, as is proved by the fact that frequently while washings from an antrum with pus will produce a positive culture, the washings from the opposite side will be reported negative.

REPORT OF CASES

CASE 1.—B. N., a boy, aged 5, was admitted to the Children's Hospital with a history of generalized edema and oliguria of ten days' duration, preceded by a cold in the head. The past history was unimportant, as was the family history. The attending physician reported no abnormalities except generalized edema. Examination of the nose and throat revealed palpable posterior cervical glands on the right side; purulent discharge in the right nares; swollen turbinates on the right side, and a straight septum. The left side was clear with good air space and no pus. The tonsils were of moderate size and cryptic; the anterior pillars were red. The posterior pharynx was normal except for a small amount of pus in the postnasal space. A roentgenogram of the sinuses was negative. The child weighed 53 pounds (24 Kg.). The blood pressure was 120 systolic and 84 diastolic. Urinalysis showed: albumin, 2 plus; hyaline casts, and no pus or red cells. The nonprotein nitrogen was 50 mg. per hundred cubic centimeters of blood. The phenolsulphonphthalein excretion was 80 per cent in two hours. The patient was placed on a salt-free diet, and fluids were restricted. Both antrums were irrigated under local anesthesia. A viscous fluid was obtained from the right side; irrigation of the left side returned clear fluid. Microscopic examination of the fluid showed pus cells, and streptococci were obtained in a culture from the right side. Microscopic examination and culture from the left side were both negative. Neosilvol and ephedrine nose drops were administered, and one week later marked improvement was noted, but some edema still remained. The right antrum was again irrigated, and a small amount of turbid fluid obtained. Three days later, the edema was entirely gone. The weight was 39½ pounds (17.9 Kg.), a loss of 13½ pounds (6.1 Kg.) in two weeks. The urinary output was normal; there was a trace of albumin, but no casts. Nasal examination gave negative results. The patient was dismissed, and the follow-up study has continued for one year with no recurrence.

A sinus infection was evidently the etiologic factor in this case.

CASE 2.—R. B., a boy, aged 3½ years, was admitted to the hospital with a history of scarlet fever at 1½ years of age, which was followed by hematuria and generalized edema. He had never been free from edema since the onset. There was also a history of a succession of subcutaneous abscesses of the leg, thigh and buttocks following soon after the attack of scarlet fever. His history otherwise was unimportant, as was the family history. The attending physician described the patient as being in poor condition and with no physical abnormalities other than generalized edema. Examination of the nose and throat revealed small, buried tonsils, red pharynx, good nasal space and no pus or polyps in either nares. There was no cervical glandular enlargement. Urinalysis gave the following results: albumin, 4 plus; many hyaline, but few granular casts; occasional red blood cells, and a few white blood cells. The phenolsulphonphthalein excretion was 85 per cent. There was 32.4 mg. of nonprotein nitrogen per hundred cubic centimeters of blood. The blood pressure was 114 systolic and 86 diastolic. The patient was placed on a high protein diet with salt and fluids restricted. Thyroid was administered to increase the metabolism. Some improvement was noticed for two weeks, when, with slight redness of the tonsils, marked edema again

returned. The condition remained practically unchanged for the next four months, during which time tonsillectomy and adenoidectomy were performed, with no change in the condition. Roentgen examination of the sinuses was repeated, and slight density of both antrums reported. Examination of the nose gave negative results. The antrums were irrigated, and a small amount of microscopic pus obtained from both sides. The culture revealed *Staphylococcus aureus*. Slight improvement followed.

Three weeks later, a bilateral window operation was performed under ether anesthesia. Five days later, the patient developed an acute infection of the upper respiratory tract, which eventually led to bronchopneumonia, and death occurred on the seventh day. The anatomic diagnosis was lipoid nephrosis and bronchopneumonia. Evidently, the nephrosis, in this case, developed secondarily to an attack of acute glomerular nephritis, which was associated with the scarlet fever. The sinusitis that developed no doubt exaggerated the symptoms but was certainly not the primary etiologic factor. The window operation on the antrum and the result emphasize the marked susceptibility of these patients to infection and their poor reaction to operative procedures.

Case 3.—C. G., a boy, aged 7, was admitted to the hospital with a history of generalized edema and oliguria of three days' duration. There was a history of a similar attack three months prior to admission, recovery taking place without treatment. In both attacks, the onset was sudden, with no record of a preceding infection of the upper respiratory tract. The boy had had scarlet fever at 1½ years of age. The attending physician reported no abnormalities except generalized edema. Examination of the nose and throat revealed palpable anterior cervical glands on both sides, no pus in the nose, normal appearing turbinates, good breathing space and large and chronically infected tonsils. A roentgenogram of the sinuses was negative.

Urinalysis showed a trace of albumin, no casts and no pus or red cells. The nonprotein nitrogen was 54 mg. per hundred cubic centimeters of blood. The phenolsulphonphthalein excretion was 60 per cent. The blood pressure was 120 systolic and 86 diastolic.

The patient was placed on salt-free, low protein diet, and the intake of fluids was restricted. He was discharged improved in one month, adenotonsillectomy being recommended for a future date.

During the next year, the patient was admitted to the hospital four times. Each time the edema was preceded by an acute infection of the upper respiratory tract. Subsidence took place, usually within a month, as a result of treatment for the respiratory infection and restriction of the diet. The albumin had increased in amount, and granular casts appeared in large numbers. Roentgenograms of the sinuses were consistently negative, and laboratory observations were unchanged from the first report.

Two years after the first admission, the nose and throat service reported the first evidence of a possible infection of the sinuses when mucopus was observed in the middle meatus of both sides. Both antrums were irrigated, and the washings were clear. No microscopic examination was made, nor was a culture taken. However, the edema rapidly subsided and was practically gone two days later. The patient was sent home with instructions for nasal treatment, and in a few weeks tonsillectomy and adenoidectomy were performed.

Four months later, the patient was again admitted with edema of the face and extremities, preceded by a cold in the head. Both antrums were irrigated, and a small amount of pus was obtained from both sides. A mixed culture of staphylo-

coccus and streptococcus was obtained. Marked improvement took place, and the patient was dismissed a few days later.

In the next two months, he was admitted twice with edema preceded by an acute infection of the upper respiratory tract. The antrums were irrigated both times, but no pus was obtained. The edema subsided as the infection cleared up. In the next eight months, he was admitted four times, each admission being preceded by an infection of the upper respiratory tract, the edema disappearing as the infection subsided. The antrums were not irrigated.

A year and a half later, the patient was again admitted with edema. A purulent discharge was seen in the left middle meatus. A roentgenogram of the sinuses was negative. The patient was placed on a restricted diet, but no improvement was noted.

Both antrums were irrigated; the washings were slightly turbid. Microscopic examination revealed many polymorphonuclear leukocytes and lymphocytes in the left side, and culture showed *Staphylococcus aureus* from that side. Microscopic examination and culture from the right side gave negative results.

Definite improvement was noted two days later, and the next recurrence occurred eight months later. The laboratory report at this time, about four years after the first admission, was: nonprotein nitrogen, 65 mg. per hundred cubic centimeters; urea nitrogen, 30 mg.; cholesterol, 250 mg.; creatinine, 2.5 mg.; total protein, 6.9 mg.; albumin, 2.2 mg.

Urinalysis showed albumin, 3 plus, and many hyaline casts.

Roentgenograms of the sinuses were reported as showing a slight density on both sides. Both antrums were irrigated, and *Staphylococcus albus* was obtained from the right side; the left was negative. There was definite improvement for only two days. The antrums were again washed, and a thick tenacious fluid was obtained from the right side. The culture again showed *Staphylococcus albus.* Edema of the face and extremities persisted, and no improvement was noted. Seven days later the procedure was repeated, and a culture was sent to the laboratory for preparation of an autogenous vaccine. The condition remained the same under a diet high in protein and restricted as to fluid and salt, with the administration of desiccated thyroid to increase the protein metabolism.

The administration of autogenous vaccine was commenced, but was soon discontinued because of poor response.

The patient's condition gradually became worse, and death occurred four years after the first admission. The anatomic diagnosis was: lipoidal nephritis, chronic diffuse nephritis, edema and congestion of the lungs and acute bronchitis.

The mucous membrane from the antrums was edematous and infiltrated with round cells, a few polymorphonuclears and eosinophils. Colon bacilli predominated so much in the culture from the sinuses that it was impossible to isolate other organisms. Culture from the abdominal fluid showed *Streptococcus viridans.*

It is evident that in this case there was a direct association between the attacks of infection of the upper respiratory tract and the attacks of edema. It is also evident that a sinusitis did occur at times and that treatment of the sinusitis in the early stages resulted in almost instant subsidence of the edema. However, there is no evidence that chronic sinusitis was the underlying etiologic factor.

CASE 4.—T., a boy, aged 3½ years, was admitted to the hospital with edema of the face and legs and oliguria. There was no history of an acute infection of the respiratory tract preceding admission. He had had, however, a series of abscesses of the legs and thighs. His past history was otherwise unimportant. Examination by the attending physician revealed an abscess of the thigh and generalized edema.

The laboratory examination showed: albumin, 2 plus, and hyaline and granular casts in the urine; nonprotein nitrogen, 25 mg., and phenolsulphonphthalein excretion, 60 per cent. The blood was normal.

Improvement took place after drainage of the abscess, together with restriction of the ingestion of salt and fluid and enough protein in the diet for maintenance.

In the next year, the patient was admitted four times with edema and a scanty output of urine. The first time he also had varicella; the second time, empyema; the third time, acute pharyngitis and tonsillitis, and the fourth time, acute nasopharyngitis.

On roentgen examination, the antrums were reported dense and were irrigated. The tonsils and adenoids were removed after the fourth admission. The results of the irrigation were negative, and no improvement was noted.

Autogenous vaccine was prepared from a nasal culture and was given, with no benefit. Stock vaccine was then given, following which the nasal discharge cleared up and the edema improved slightly. The general condition remained practically unchanged, except for slight exacerbation for some time when, following a slight infection of the upper respiratory tract, the antrums were reported slightly dense, and irrigation was performed. Pus cells were found in the right side, and the culture revealed a mixed staphylococcus and streptococcus.

Two weeks following the irrigation, the edema had entirely subsided, and no recurrence had been noted up to the time this article was written, three years after the original admission and ten months since irrigation of the antrum.

In this case, the development of a sinus infection evidently was an exciting factor, but not the original primary cause of nephrosis.

CONCLUSIONS

1. Edema and swelling of the nasal and sinus mucous membrane, occurring as the result of nephrosis, may lower the resistance and thus predispose to sinus infection.

2. Disease of the paranasal sinuses is a frequent complication of nephrosis and may aggravate the existing condition.

3. Disease of the paranasal sinuses may be an important etiologic factor in the production of nephrosis.

SPASM OF THE AUDITORY ACCOMMODATIVE MECHANISM

A FUNCTIONAL BASIS OF EAR-DRUM RETRACTION AND OF DEAFNESS FOR LOW TONES *

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It has been assumed in the past by otologists that retraction of the tympanic membrane is due to one of two causes: first, and most frequently, to catarrh of the eustachian tube and to rarefaction of the air in the tympanic cavity; second, to adhesions of the tympanic membrane to the promontory and the inner wall of the middle ear. It has generally been assumed that all of the cases presenting this sign, retraction of the ear-drum, which were not due to the first cause and which did not respond to inflation would be found to be due to the second cause, especially when associated with chronic progressive deafness.

A prolonged study of a large series of cases presenting retraction of the ear-drum, none of which was relieved by politzerization or by other forms of inflation, has revealed a hitherto unrecognized and undescribed functional type of ear-drum retraction, which is associated with deafness, or rather deafening, for the lower tones. The study has also revealed a method of treatment for this condition.

The starting point of the investigation was the observation that many cases showing a retraction of the ear-drum that would not respond to inflation presented normal or atrophic nasal eustachian ostia and eustachian tubes that were apparently patent to the catheter. On incision of the retracted drum, the patency of the eustachian tube was fully confirmed in two manners: first, it was found that air could be blown through the tube and into the middle ear by any method of inflation and quite freely, depending on the extent of the incision; second, it was found that medicated powders could readily be blown into the nose by way of the ear and the eustachian tube.

A theoretical consideration of the possible etiology of retraction of the tympanic membrane led to suspicion of the possible rôle of the ossicular chain and the muscles thereto attached, the tensor tympani and the stapedius. On the assumption that there might exist in these cases a spasm of the muscles involved, some method of relief of such presupposed spasm was sought. Various methods were tried, but it was not

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until the use of the negative pole of the galvanic current was applied that success was obtained in the relief of these cases of spastic retraction of the drum.

After one or several applications of the negative pole of the galvanic current to the ear, it was found that the previously immobile drums in these cases became freely mobile, responding even to gentle valsalva inflation. The hearing defect in these cases, ranging from 10 to 30 decibels (sensation units) in the frequencies 30 to 1,000 (together with a slight lowering of hearing for higher frequencies) was either reduced or totally cleared up.

To complete the chain of evidence that would verify our hypothesis and prove it to be a fact, a quest was made for a series of cases in which a reflex origin of the muscles of the accommodative mechanism of the ear could be proved. A number of such cases were found, the clearest and most interesting of which was a case of unilateral retraction associated with diplacusis and reduced hearing for low tones, but with no inflammatory changes in the ear involved, secondary to an ipsolateral antrum infection with lower-half headache and trigeminal neuralgia. In this case, relief of the retraction of the drum and of the diplacusis and hearing defect was obtained in three manners successively: by galvanization, by cocainization of the sphenopalatine ganglion and by relief of the antral condition.

As to the determined and suspected causes of spasm of the tensor tympani and stapedius muscles and the resultant spasm of the accommodative mechanism of the ear, a number of interesting observations have been made, and the literature supplies us with some pathologic data. Spiesman [1] showed that the fat deposits normally found in the tensor tympani muscle are diminished in otosclerosis, indicating an overaction and hypertrophy of the muscle.

Concerning spasms due to remote reflexes little need be said here, except that in this type of case the therapy must be directed at the remote cause primarily and at the spasm itself secondarily. Direct irritation occurs in the tensor tympani and stapedius muscle as a result of an impairment of their circulation such as has been noted in chronic progressive deafness (either an arterial and general circulatory defects such as those described by me [2] as characteristic of the disease and as a fundamental pathologic factor in it, or the probably secondary venous stasis described by Witmaack and Manasse as characteristic of otosclerosis); this irritation apparently gives rise to a spasm of the muscles.

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1. Spiesman: Ueber die Fetteinlagerungen im Musculus tensor tympani, Monatschr. f. Ohrenh. **61**:971, 1927.

2. Josephson, E. M.: Laryngoscope **39**:40 (Jan.) 1929.

On the degree of irritation and vascular impairment appears to depend the presence or absence of impairment of hearing for low tones in chronic progressive deafness.

There is ample evidence at hand to indicate that in these cases the relief of the spasm of the accommodative muscles through the administration of galvanism is obtained through the influence of the current on the vascularity of the middle ear, as well as through its direct effect on the muscle spasm. It should be noted that the hearing for higher pitches is also improved, in lesser degree, by this therapy.

The fact that, as a result of the spasm of the accommodative mechanism of the ear, the low tones are diminished in intensity and impaired, whereas the sensitivity of the auditory nerve is unimpaired, as is rendered manifest by preservation of bone conduction as well as by improvement of air conduction on myringotomy, throws interesting light on the function of the accommodative mechanism of the ear. Retraction of the drum diminishes the intensity of low tones. This protective retraction (which serves to protect the mechanism of the inner ear from trauma due to the high energy value of the intense, low tones) in order to be effective as a protective device, must be a prompt reflex, the afferent nerve arc of which must reside in the innervation of the tympanic membrane and of the ossicular, accommodative mechanism; for if the afferent impulse should pass through the inner ear and the auditory nerve, in such a manner that an intense sound might first shatter the mechanism of the inner ear, the tympanic retraction device would not serve to protect the mechanism of the inner ear. On the other hand, that the accommodative mechanism of the ear does influence the intensity of sound stimuli reaching the inner ear, necessitates the recognition of a hitherto unappreciated factor in the sensation of hearing, the possible rôle of the sensation, deep and otherwise, of the accommodative mechanism of the middle ear as a gage of the intensity of sound and the summation of this sensation into the perception of sound and its qualities. West and Barlow,[3] in their hypotheses as to the mechanism of the central beat note, predicated "a kinesthetic sensation that the subject interprets as an increase in volume" which, they supposed, originates from a stimulation of the vestibule which causes an increase in tonus of the muscles of the middle ear. But it is obvious that such a modus operandi would not serve to protect the mechanism of the inner ear from the trauma of overloud sounds. On the other hand, their discussion of "overflow" of nerve excitation indicates the far higher probability of the origin of the accommodative ear reflex by way of afferent nerve fibers other than those supplying the inner ear, a hypothesis which I have advanced and partly proved.

3. West and Barlow: The Neuromuscular Mechanism of Hearing, Arch. Otolaryng. **9**:645 (June) 1929.

Explanation can be offered for the loss of conduction of low tones when the ear-drum is fully retracted by overaction or spasm of the tensor tympani muscle, in spite of the fact that when the drum is incised, with no apparent change in the position of the ossicular apparatus, low tones become promptly audible in diminished intensity. The intact tympanic membrane must be set in vibration by the sound waves in order that they may be transmitted to the middle ear and thence to the inner ear. The rigidly retracted drum, with its altered vibrating area elasticity and natural frequency, does not respond in normal measure to the lower frequencies, and consequently those notes are not transmitted to the middle ear. When the ear-drum is opened, the low tones are directly transmitted to the middle ear and thence to the inner ear. (The fallacy of the assumption that the ossicular apparatus is the essential factor in the transmission of sound to the inner ear is made obvious by the numerous cases in which there is fairly normal hearing though the drums are destroyed.) When the spasm of the accommodative mechanism of the middle ear is relieved, the low tones are again transmitted by the mobile drum. The question as to whether sound waves are transmitted to the inner ear by way of the round window is a very pertinent one in this connection.

A number of the academic phases of the matters here presented are now under investigation, and will be reported on in the future.

SUMMARY

There exists a definite type of deafness for low tones associated with retraction of the tympanic membrane which will not respond to inflation and which is not associated with any gross pathologic process of the mechanism of the middle ear. Both the deafness for low tones and the retraction of the tympanic membrane are relieved by galvanization of the ear. The improvement is due to relief of the spasm of the muscles of the accommodative mechanism of the middle ear.

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FURTHER OBSERVATIONS ON AGE VARIATIONS IN AUDITORY ACUITY *

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In an earlier paper [1] is presented a statistical study of the records secured by testing the auditory acuity of 353 patients from the medical and surgical departments of the Johns Hopkins Hospital. The results of the study indicate that if the averages of the thresholds are computed, there is a distinct loss of acuity in each successive decade for tones above 512 double vibrations, but the averages of the thresholds for tones of lower pitch are fairly constant for all patients regardless of age. The statement was made that "it is hoped that the information thus secured will make possible the statistical analysis of the auditory defects associated with cerebral arteriosclerosis, hypertension, hypotension, syphilis, nephritis, disturbances of the ductless glands, the various diseases of the blood, lesions of the central nervous system and other conditions."

In this paper the age variations in auditory acuity in a group of 468 patients are presented together with a statistical analysis of the effects of arteriosclerosis, hypertension, chronic cardiac conditions, syphilis and various malignant conditions on auditory acuity.

The conditions of the tests were identical with those of the former study, with two exceptions. The hearing tests included in this paper were made by five examiners [2] rather than by two, and many of the patients with syphilis were sent from the medical dispensary direct to the laboratory for hearing tests without admission to the hospital wards. None of the records included was from a patient who admitted loss of acuity. All were tested with the same 1A Western Electric audiometer. The tests were conducted either in the wards or in a small room which was far from being sound-proof. Neither was ideal for conducting hearing tests. Disturbing noises often interrupted the tests, but repeated checks by different examiners on the same patients, in the ward or the testing room, showed that the results were consistently within the limits of the attention factors of the patient.

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* From the Otological Research Laboratory, Johns Hopkins University.

1. Bunch, C. C.: Age Variations in Auditory Acuity, Arch. Otolaryng. **9**:625 (June) 1929.

2. The tests were conducted by Dr. J. J. Belemer, Miss Harriet Auld, Mr. W. W. Young, Mr. C. E. Balfour and me. Miss Auld also aided in the preparation and compilation of the data presented in this study.

Assuming the effects of age, shown in the previous study, to be a constant, the audiograms were divided into decade groups and redivided according to the clinical diagnosis. The results were calculated on this basis. This classification necessitated some overlapping, as the diagnosis in some cases included more than one of the conditions studied. If a record belonged in two or more groups, it was tabulated in each.

Chart 1 shows the "scattergram" for the acuity thresholds of forty-three patients between 20 and 30 years of age. The numbers in the vertical column correspond to the intensity readings in sensation units taken from the audiograms. Those in the horizontal column indicate the

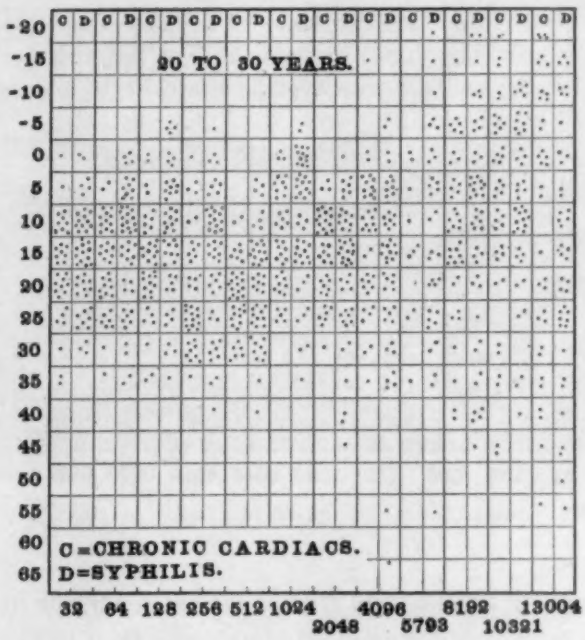

Chart 1.—Scattergram showing the acuity thresholds of forty-three patients between 20 and 30 years of age. In this and the following charts, loss of hearing is represented in sensation units on the vertical scale at the left. The figures along the bottom of the chart indicate the tones.

pitch of the tones for which thresholds were determined. Each dot on the scattergram represents the threshold for one ear. Arteriosclerosis, hypertension and malignant conditions are infrequent at this age, and consequently the scattergram contains only the thresholds for patients with chronic cardiac conditions (nineteen cases), in the columns lettered *C*, and with syphilis (twenty-four cases), in the columns lettered *D*.

It will be noted that the scattering of the dots representing the thresholds is uniform for all tones under 1,024 double vibrations, and is

similar for both types of cases. The area of scattering becomes somewhat wider at 2,048 double vibrations and increases for tones of higher pitch. There is also a considerable number of dots above the indicated normal (marked 0 on the chart) for these high pitched tones. Almost all the subjects could hear the tone of 13,004 double vibrations, but the scattering was wide. Only fourteen patients with chronic cardiac conditions and fourteen with syphilis could hear the tone of 16,384 double vibrations at the maximum intensity of the audiometer, so the thresholds for this tone are not tabulated on the scattergram. The scattering of thresholds for patients with syphilis is similar to the scattering for those with chronic cardiac conditions.

The averages for these thresholds, calculated and plotted to the audiometric scale, are shown in chart 2. The fact that the line of the averages for the patients with syphilis lies close to and several times

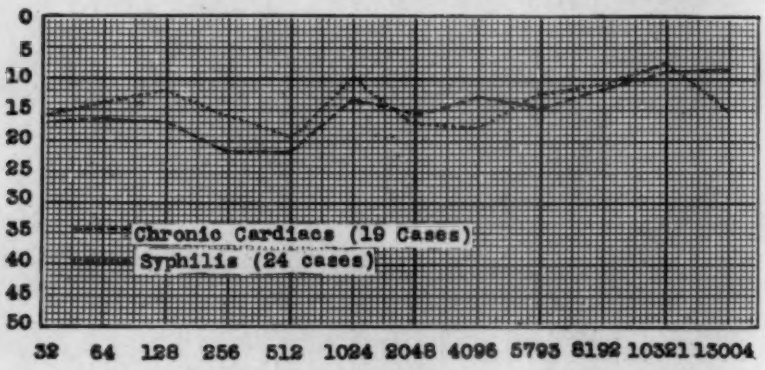

Chart 2.—Averages for acuity thresholds shown in chart 1, plotted to the audiometric scale.

crosses that for patients with chronic cardiac conditions indicates that there is no marked difference in the averages for these two groups at this decade.

Chart 3 shows the scattergram for patients between 30 and 40 years of age. In this group are twenty-eight patients with chronic cardiac conditions, forty-three with syphilis, thirteen with hypertension, eight with arteriosclerosis and twenty-two with malignant conditions. The scattering of the thresholds for these patients is similar to that for patients in the 20 year group; i. e., it is uniform for tones below 1,024 double vibrations for all five conditions, and it becomes more marked for tones of higher pitch. It should be noted that on all scattergrams there are fewer thresholds charted for 5,793 than for 4,096 and 8,192 double vibrations. This is because the tone of 5,793 double vibrations was not used as a routine stimulus by all examiners.

Chart 4 shows the calculated averages for the thresholds shown in chart 3, plotted to the audiometric scale. It will be noted that the curves lie close together on the chart and cross each other repeatedly. In view of the fact that there are but eight cases of arteriosclerosis to be compared with forty-three cases of syphilis, it is significant that the averages are so similar.

Chart 5 shows the scattergram of the thresholds for patients between 40 and 50 years of age: seventeen with arteriosclerosis, twenty-four with hypertension, thirty-eight with chronic cardiac conditions, forty-five with syphilis and twenty-nine with malignant conditions. Again, the thresh-

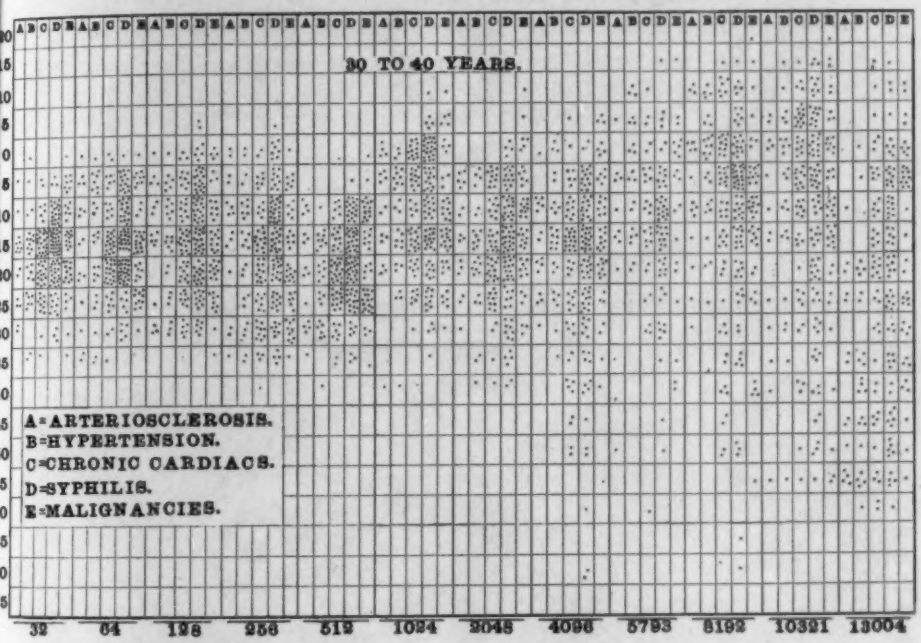

Chart 3.—Scattergram showing the acuity thresholds of patients between 30 and 40 years of age.

olds below 1,024 double vibrations are scattered uniformly for all groups. A significant difference between chart 5 and charts 1 and 3 is apparent. Few thresholds for tones of high pitch lie above the indicated normal (marked 0 on the scattergram) in the 40 year group. So few in the 40 year group heard the tone of 13,004 double vibrations that it was not tabulated.

Chart 6 shows the averages of these thresholds plotted to the audiometric scale. Here again, all the curves cross frequently. No curve indicates significantly better hearing unless it is that for syphilis. As stated before, however, many of the patients with syphilis were sent

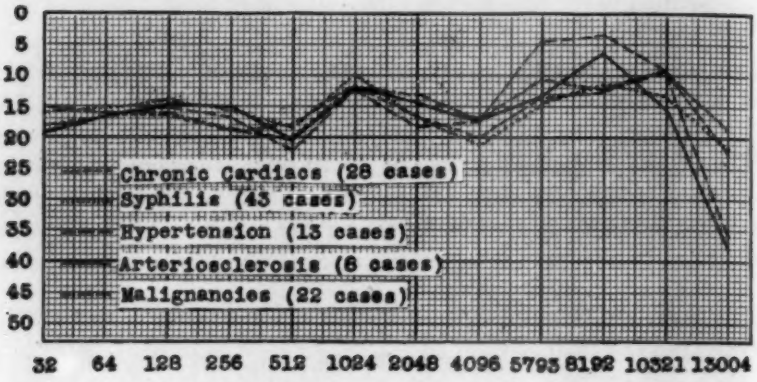

Chart 4.—Averages for the thresholds shown in chart 3, plotted to the audiometric scale.

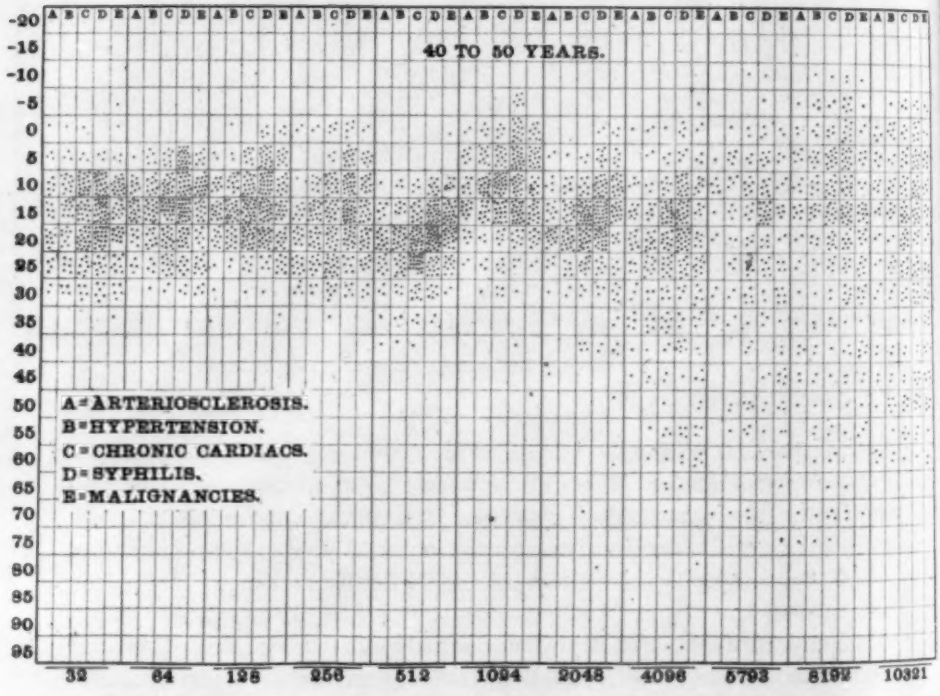

Chart 5.—Scattergram showing the acuity thresholds of patients between the ages of 40 and 50 years.

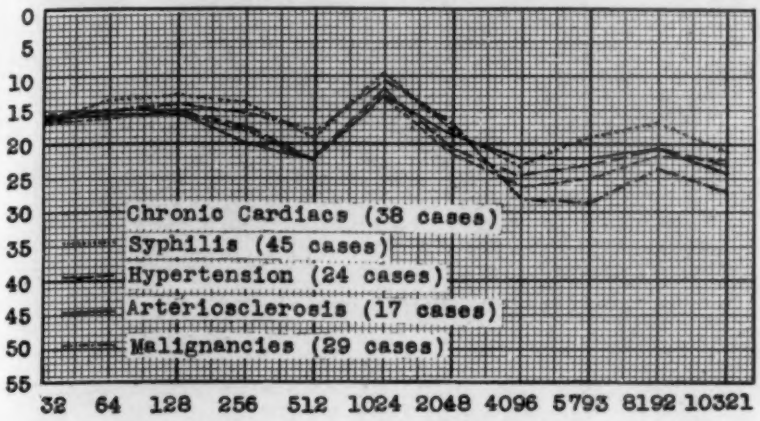

Chart 6.—Averages of the thresholds shown in chart 5, plotted to the audiometric scale.

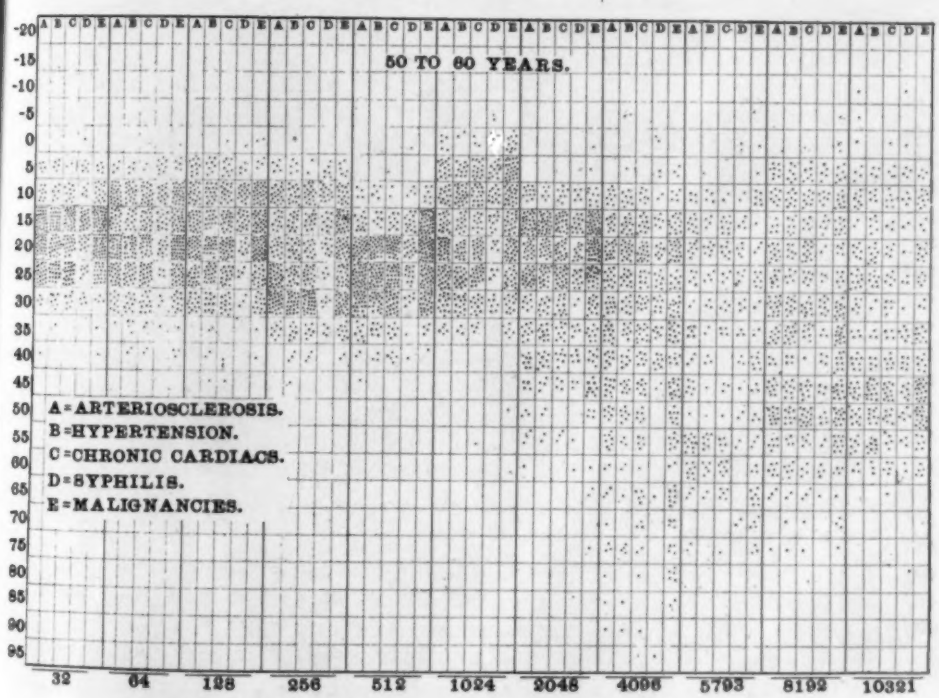

Chart 7.—Scattergram for patients between 50 and 60 years of age.

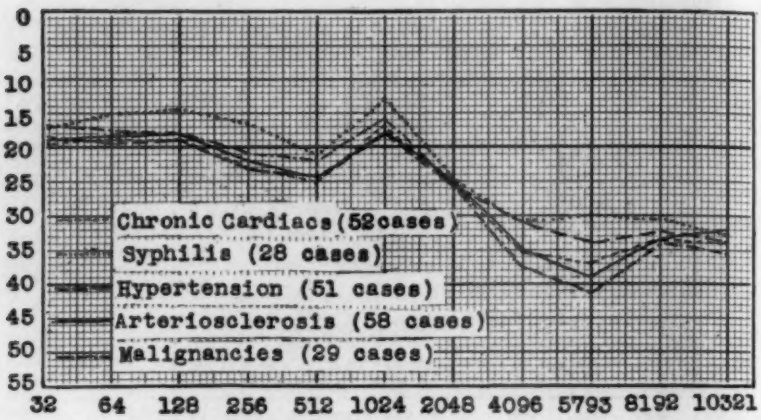

Chart 8.—Averages of the thresholds shown in chart 7, plotted to the audiometric scale.

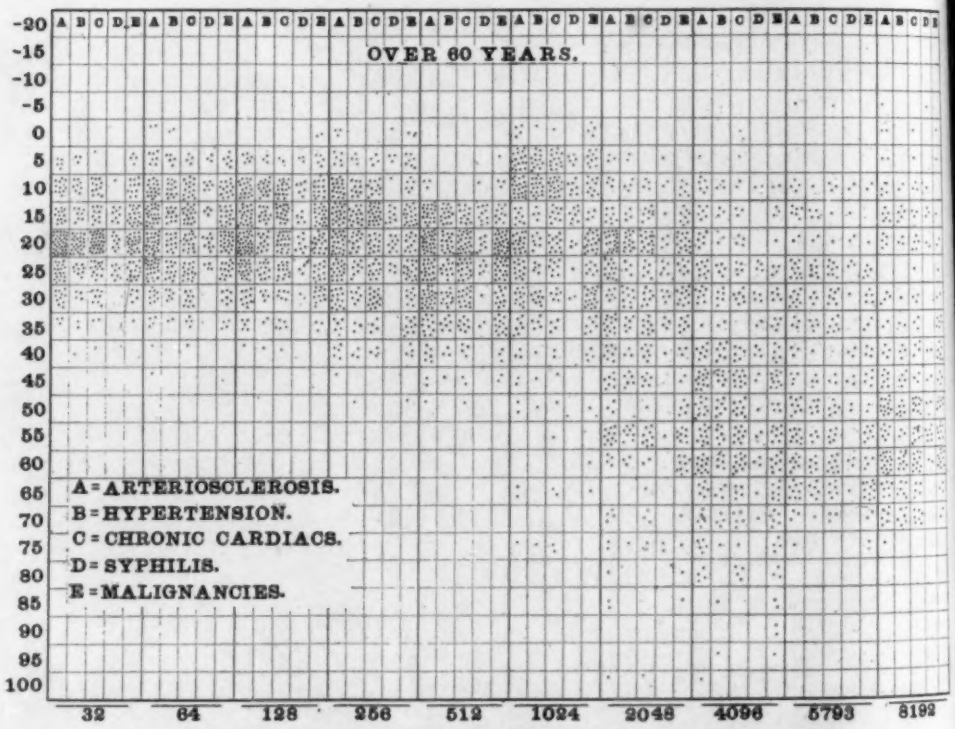
OVER 60 YEARS.
A = ARTERIOSCLEROSIS.
B = HYPERTENSION.
C = CHRONIC CARDIACS.
D = SYPHILIS.
E = MALIGNANCIES.
32 64 128 256 512 1024 2048 4096 5793 8192

Chart 9.—Scattergram showing the acuity thresholds of patients over 60 years of age.

directly to the laboratory for hearing tests without being admitted to the hospital. It may be that patients ill enough for hospitalization are not in condition to listen with utmost attention to auditory tests. Data are not here available to support this contention. This is given only as a possible explanation.

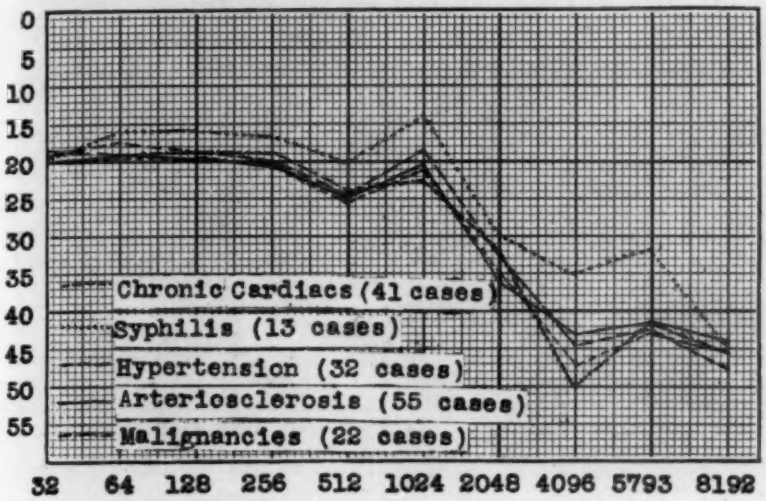

Chart 10.—Averages of the thresholds shown in chart 9.

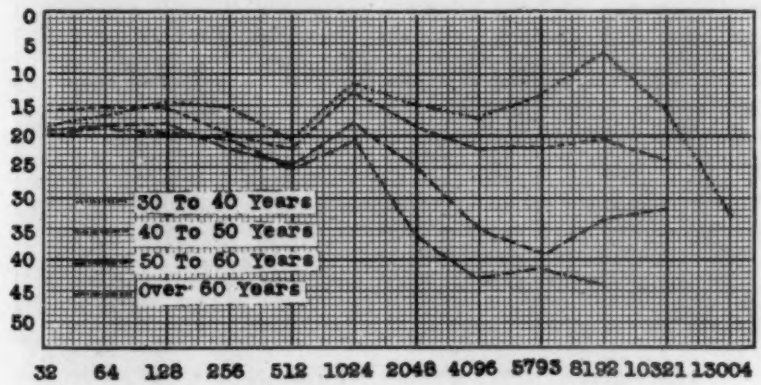

Chart 11.—Averages of the thresholds of patients with arteriosclerosis.

Chart 7 shows the scattergram for patients between 50 and 60 years of age: fifty-eight with arteriosclerosis, fifty-one with hypertension, fifty-two with chronic cardiac conditions, twenty-eight with syphilis and twenty-nine with malignant conditions. The conditions noted in the description of chart 5 are more evident in chart 7. The thresholds for low tones are uniform, but the thresholds for the high tones are much

lower on the chart. Chart 8 shows the averages of the thresholds of the 50 year group plotted to the audiometric scale. As in chart 6, all the averages cross frequently, except those for patients with syphilis. The explanation given before may possibly be the reason for the better hearing indicated for patients with syphilis.

Chart 9 shows the scattergram for patients over 60 years of age: fifty-five with arteriosclerosis, thirty-two with hypertension, forty-one

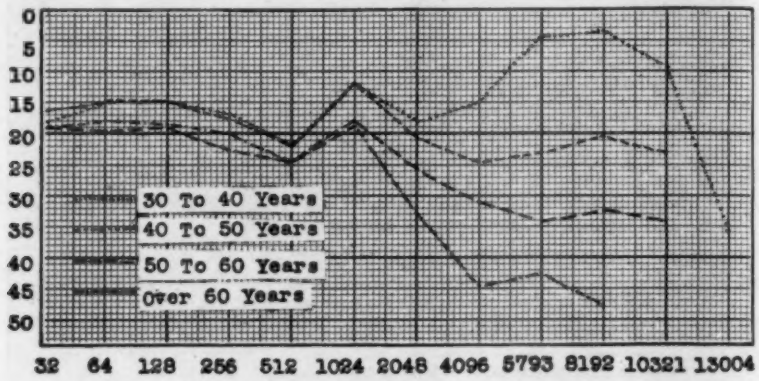

Chart 12.—Averages of the thresholds of patients with hypertension.

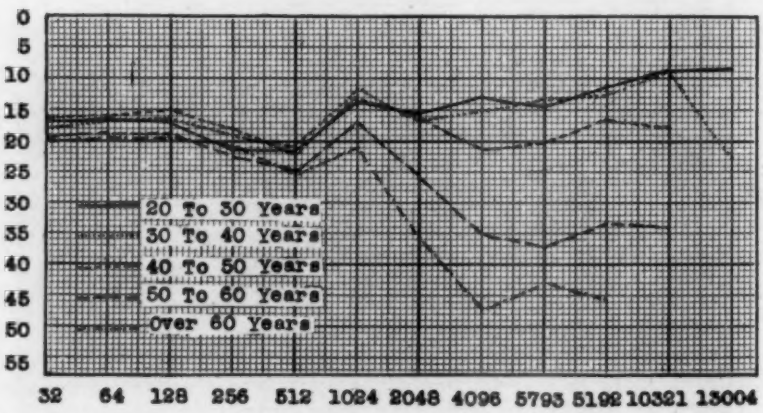

Chart 13.—Averages of the thresholds of patients with chronic cardiac disease.

with chronic cardiac conditions, thirteen with syphilis and twenty-two with malignant conditions. Few were able to hear the tone of 10,321 double vibrations, so it was not tabulated. Chart 10 shows the calculated averages of the thresholds plotted to the audiometric scale. The average for patients with syphilis is, as before, definitely higher than those for patients with the other conditions tabulated.

Charts 11, 12, 13, 14 and 15 show the averages for the thresholds of the patients with arteriosclerosis, hypertension, chronic cardiac conditions, syphilis and malignant conditions, respectively, plotted to the audiometric scale. It will be noted in each that tones below 1,024 double vibrations are heard almost equally well in all decades by patients in whom the diagnosis included one or more of the five conditions studied.

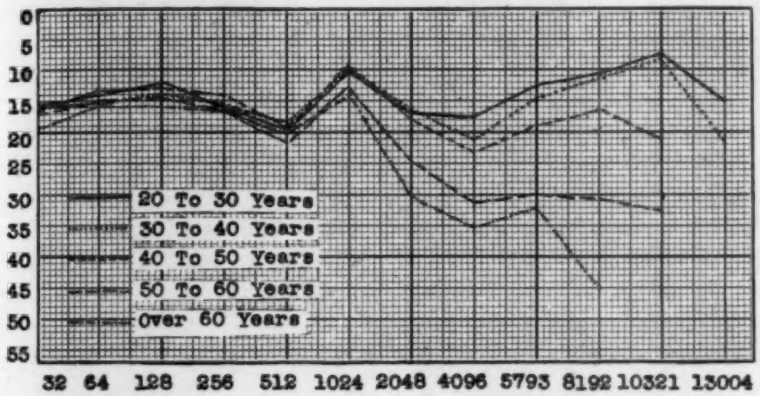

Chart 14.—Averages of the thresholds of patients with syphilis.

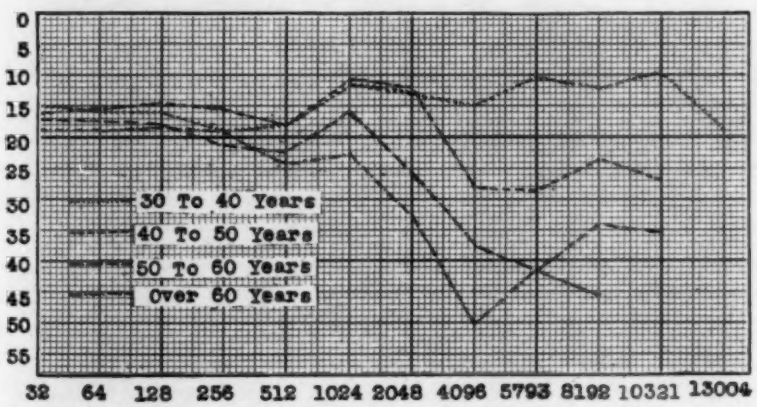

Chart 15.—Averages of the thresholds of patients with malignant conditions.

For tones of higher pitch, there is a constant loss for each disease, increasing with advancing age and for tones of higher pitch.

SUMMARY AND COMMENT

The loss in acuity for tones of high pitch with advancing age, which was shown in an earlier study, is a constant factor in patients in whom a clinical diagnosis of arteriosclerosis, hypertension, chronic cardiac conditions, syphilis or malignant conditions has been made. No one of these

more or less generalized systemic conditions can be considered responsible for this loss.

Arteriosclerosis is said to be a constant factor which increases with advancing age. If this is true, then it parallels the loss shown in this study and in the one previously presented. The observations in the present study seem opposed to this contention, as patients in all decades for whom a definite clinical diagnosis of arteriosclerosis has been made do not show a greater loss than those for whom such a diagnosis was not made.

OSTEOMYELITIS ASSOCIATED WITH FRONTAL SINUSITIS

VALUE OF PRESERVING ANTERIOR WALL WITH ATTACHED PERIOSTEUM: REPORT OF CASES *

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LOS ANGELES

The title of this paper shows my reason for reporting the two cases. Osteomyelitis associated with frontal sinusitis is of common occurrence, and these cases would not be reported if it were not for the fact that gradual healing of the untouched inflamed bone is demonstrated. The "fulminating" fatal type of case occurs too frequently. For example, in Bulson's review of the literature [1] fifty-five cases with thirty-seven deaths are recorded. Twenty-eight of the deaths occurred as complications of chronic infection of the frontal sinus, and—this is important to emphasize—twenty of these twenty-eight cases followed radical operative procedures. One can only guess how many more cases have terminated fatally and have not been reported. Three years ago I reported to the American Laryngological Association a case in which meningitis rapidly developed from an acute frontal osteomyelitis. The patient recovered, although nothing was done except to drain the frontal sinus; it was believed that a fatal result was inevitable. In my paper I stated that my surgical work had nothing to do with the recovery, unless it was due to the fact that I did so little, my therapy consisting of drainage through the floor of the frontal sinus. Several weeks later a secondary radical operation was done for the removal of sequestrums. This case caused me to study osteomyelitis, not only that which occurs in laryngologic practice, but that which occurs in general orthopedic practice, and I became convinced that laryngologists were doing too much surgical work in cases of osteomyelitis and thereby were increasing the chances for its rapid spread.

That inflammation of the bony walls of the frontal sinus occasionally occurs and ceases without operative measures is well known. I have come to believe that it is not generally realized that such healing is hindered by meddlesome surgical intervention, particularly by stripping

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* Read at the Meeting of the American Laryngological, Rhinological and Otological Society, Atlantic City, N. J., May, 1930.

1. Bulson, A. E., Jr.: Osteomyelitis of Frontal Bone as Complication of Frontal Sinusitis, J. A. M. A. **86**:246 (Jan. 23) 1926.

of the periosteum, the real mesentery that feeds the bone with nourishing blood. I believe that when there is an acute inflammation of the bony wall of the sinus caused by acute or chronic suppuration of its lining membrane, the inflamed bone should be let alone. Nature is endeavoring to stop the process, and will do her best if left alone; otherwise the extension of the inflammation of the bone is hastened until an incurable so-called "fulminating" osteomyelitis ushers in death by meningitis or abscess of the brain. Drainage of the frontal sinus by an opening in its floor during an acute inflammation or an acute exacerbation of a chronic inflammation offers surgical relief and gives nature a chance. When the acute symptoms subside, there is time enough for whatever radical measures are needed; in other words, a two-stage operation is indicated. It is conceded that a swollen, tender frontal sinus, with edema of the eyelids, fever and pain from an acute inflammation of the sinus simply requires an opening for drainage, but this should be obtained without stripping the periosteum of the anterior wall. I am also persuaded that an acute lighting up of a chronic condition of the frontal sinus should be treated surgically in the same way. Heretofore, the common practice in all chronic cases has been to perform thorough exenteration of the frontal sinus, only a few operators (notably Robert Lynch) having taken care not to injure the anterior wall and its attached periosteum. Too many patients have died as a result of this operation. I believe that the number of deaths will be reduced considerably by drainage and by giving nature a chance; after the acute symptoms have subsided, radical measures can more safely be taken. Even then the less violence to the anterior wall and its attached periosteum, the better.

I am told by competent orthopedic surgeons that this theory conforms to the surgical procedure practiced by them during the last few years. They have gotten away from the method of complete exenteration, and as a result have fewer fatal cases due to spread of the osteomyelitis.

It is time to emphasize the extremely important part played by the periosteum that overlies and is attached to acutely inflamed bone. Otolaryngologists can learn some lessons from orthopedists. Gallie is quoted by Winnett Orr,[2] as follows:

> The periosteum is a fibrous membrane in which the blood vessels to the superficial layer of the bone ramify before entering the haversian canals, and through which pass the large nutrient vessels on their way to the medullary cavity. [Of course, in the case of the frontal bone, there are no large nutrient vessels, but innumerable small vessels.] It therefore follows that stripping of the periosteum

2. Gallie, quoted by Orr, Winnett: Osteomyelitis and Compound Fractures and Other Infected Wounds: Treatment by the Method of Drainage and Rest, St. Louis, C. V. Mosby Company, 1929.

will interfere more or less seriously with the circulation in the bone, depending upon the size and the number of the blood vessels destroyed. When this occurs in an aseptic field, as in simple fractures, no gross disturbance results, and it has been customary to suppose that no important pathological change has occurred in the portion of bone so denuded of periosteum. . . . That this is an error is witnessed by the fact that the rapidity of union of simple fractures is frequently inversely to their severity; and by the observation of Robertson and the writer [Gallie], that following the stripping of the periosteum over even small areas of the shafts of the bones of animals, coagulation of the blood in the haversian canals occurs to a considerable depth, and the lacunar cells which derive their nutrition from these blood vessels become necrotic and slowly disappear.

In the presence of infection the effect of the stripping of the periosteum is to interfere with nature's process of fighting the osteomyelitis; a rapid "fulminating" osteomyelitis is the dreaded result that occurs so often after surgical procedures on the frontal sinus. Gallie also explained how, in the presence of infection, the stripping of the periosteum destroys the superficial osteoblasts that are responsible for the regeneration of bone. He also stated that a mild septic infection is the greatest stimulant to the repair of bone.

Applied to laryngology, this suggests adapting surgical procedures to these principles; in other words, let the acutely inflamed periosteum and bony wall alone, until through drainage of the frontal sinus nature has had a chance to localize the inflammation, and to effect a cure without further surgical intervention, as in the two cases I am reporting.

REPORT OF CASES

Case 1.—*History.*—A man, aged 23, seen on Aug. 19, 1929, gave a history of severe left frontal headaches of one week's duration, following swimming. There were no other symptoms, except a slight cold in the head. The left eye had become red four days after the onset of the headache. He had experienced a similar attack of headache a year previously. He had frequent slight colds and headaches, but no chronic nasal discharge.

Nasal examination revealed a septum that was deviated to the left; the mucous membrane was hyperemic and the turbinates engorged; there was a thin serous discharge from both sides of the nose. The pharynx was slightly red; the tonsils had been well removed. There was definite tenderness over the left frontal sinus. The left conjunctiva was congested, and the eye lachrimated profusely. The temperature was normal. Transillumination revealed density of the left frontal sinus. Roentgenograms showed a definite increase of density of the left frontal sinus. The patient received daily intranasal treatment with some degree of relief, but he was never free from headache for any length of time.

On September 13, four weeks from the onset, he reported that his forehead became sore and swollen. The nasal discharge had been absent for a week. Examination showed a swelling over the left frontal sinus above the left eyebrow, extending 1¼ inches above and 1¼ inches (31.7 mm.) to the left of the midline. Headaches were not present, but there was slight tenderness of the anterior wall and of the floor of the frontal sinus. Roentgen examination showed no material change from the previous examination a month earlier.

The patient was admitted to the hospital on September 13. The temperature was 100.2 F. Examination of the blood showed: 96 per cent hemoglobin, 5,440,000 red blood cells; 12,500 leukocytes; 77 per cent polymorphonuclears, and 20 per cent lymphocytes.

Operation.—At operation on the following day (September 14), an incision was made through the left eyebrow. Care was taken not to elevate the periosteum over the anterior wall. The sinus was entered through the floor, and the opening was enlarged for good drainage. When the sinus was opened, pus was found under pressure; it was thick, yellow and without odor. The lining membrane was moderately inflamed. Culture of the pus was reported to be negative, but later *Staphylococcus aureus* was found.

Course.—On September 15, the following day, the temperatue was 101.2 F. There was a moderate amount of discharge.

On September 17, the temperature was 100.8 F.; the following day it was 99.2 F., and from then on it remained normal. The patient was discharged from the hospital in ten days in good condition, with little drainage from the sinus. The use of dressings was continued at the office. There was rapid improvement. The edema of the wall of the frontal sinus gradually decreased.

Two weeks later, on September 30, the patient reported that he had had a temperature of 101 F. on the preceding day, accompanied by a chill, and a temperature of 102 F. in the evening, but that in the morning, the temperature was normal. There was no sore throat or nasal discharge. Nothing could be found to account for the fever.

The condition of the wound was good. There was a small amount of mucopus and slight edema of the eyelid. Roentgen examination showed a slight density of the left frontal sinus; there was no evidence of bone absorption. The patient was hospitalized for observation. Examination of the blood showed: 6,600 white blood cells, 67 per cent polymorphonuclears and 29 per cent lymphocytes. A culture of the blood showed *Staphylococcus aureus* and a gram-positive diplobacillus. After five days' observation, the patient was pronounced in good condition. The temperature gradually fell from 101 to normal. Ten days later (October 16), the wound was allowed to close, as there had been a little stringy mucopus, but there was no fever, tenderness or nasal discharge. Eleven days later, the patient was discharged as cured. Three weeks later, November 19, he reported for observation. The notes taken on the history state that he had had no trouble. At no time had there been nasal secretion blown out or hawked backward. The scar under the left eyebrow was firm; there was no soreness, swelling or complaint of any sort.

On December 20, three months after the operation on the frontal sinus, roentgen examination was made in spite of the fact that the patient showed no signs or symptoms of sinus trouble, in order to see the condition of the anterior wall of the frontal sinus. The roentgenograms showed a normal frontal sinus and a distinct change in the anterior wall, resulting from the healing of the bone.

The roentgenograms taken in August, September and December show, I believe, the changes that occurred in the anterior bony wall, corresponding to the edema of the forehead. At the time of operation there had been no elevation of the periosteum over the edematous area of the frontal sinus in order to see the condition of the underlying bony plate. This fact was discussed, especially as the father of the patient had seen the roentgenogram that showed inflammatory changes of the anterior bony wall. I had also told him that a secondary operation might be required if a sequestrum formed.

I wish to report a similar case which demonstrates, I believe, the changes that occurred in the bone of the anterior wall of the frontal sinus during the period of healing.

CASE 2.—H. F., a girl, aged 7, seen on March 30, 1925, presented a swelling of the left eyelid and over the left frontal sinus of three days' duration, accompanied by a slight fever. A severe attack of acute inflammation of the nose and throat had preceded the swelling.

Examination showed that the left eyelids were greatly swollen; there was a soft edema, making it almost impossible to see the eyeball, and a slight swelling over the frontal sinus near the midline. There was no apparent symptom pointing to an abscess at any place. The eyeball was partly seen and freely movable. The left side of the nose was entirely blocked by swelling of the turbinates. It was impossible to see the middle meatus even after prolonged shrinking. Roentgen examination showed that the left frontal and ethmoidal areas and the antrum were cloudy.

Cold applications were advised, while further observation was continued. Two days later, on April 2, the condition was somewhat improved. There was less swelling; there were no chills or headache. The temperature was 100 F. A redness of the skin and firm edema were observed over the nasal process of the inferior maxilla; the eyelids were edematous, and the nasal condition was unchanged. The temperature became normal twenty-four hours later, and the swelling rapidly subsided.

Seven days later, except for a slight swelling and a very slight tenderness in the frontal region, near the midline, the patient had entirely recovered and left the hospital. For a month she was under daily observation. The acute nasal condition had subsided rapidly, with little pus at any time.

Slight swelling and tenderness over the frontal sinus recurred on May 3, four weeks after the initial onset, at the same place where marked swelling had been located while the patient was in the hospital, but there was no recurrence of the edema of the eyelids. Roentgen examination showed a small area of necrosis of the outer table of the frontal sinus in the midline and some thickening of the mucosa. Otherwise the child had entirely recovered. The temperature had been normal for three weeks; she was in excellent physical condition and had gained 6 pounds. The nasal condition had remained normal, without signs of pus. In view of the apparent recovery, without operation, it was concluded to wait a few days, during which the patient was carefully observed by myself and by Dr. W. H. Wimp, laryngologist, of Pasadena, and also by Dr. Carl Rand of Los Angeles, a neurologic surgeon. Dr. Rand's observations were negative, except that he expressed the belief that no immediate operation was indicated, in which Dr. Wimp and I concurred. The report of Dr. Rand was as follows:

"While there is little doubt in my mind that the child has a low-grade infection of the anterior wall of the frontal sinus, I would be inclined against surgical intervention at present. There will be ample time to explore this region if her symptoms recur. The neurologic examination is negative."

The slight swelling over the frontal area gradually subsided, and within three weeks it was barely noticeable.

Five months later, my history notes stated that the patient had not been seen in two months; she had been perfectly well and there were no colds or headache. She had gained in weight, and appeared in perfect health. The thickening over the frontal sinus in the midline had entirely disappeared, leaving a flatness, a

depression, even to the touch, as if the outer table had been slightly eroded and healed. Nasal breathing was good on each side; there were no signs of an intranasal pathologic condition. The tonsils and adenoids were then removed. Six months later (April 4, 1926), one year after the acute condition of the frontal sinus occurred, the patient was brought in for observation and was normal. The slight depression could still be made out by careful palpation.

During the past three and one-half years I have often met the father and mother and at times the child, so that to date I know that there has been no recurrence.

There was much discussion at the onset of the child's condition, regarding the possibility of relief without surgical intervention and particularly regarding the scar that would result from opening the swelling of the frontal area and curetting the frontal plate which the roentgenogram showed to be diseased. But although I was ready to operate, daily observation showed all the signs and symptoms improved, and a final recovery resulted without a surgical procedure. Therefore I am able to report a case in which active inflammation of the frontal bone subsided without surgical intervention. Roentgenograms taken on May 1, May 5, June 2 and Oct. 12, 1925, will show, I think, the area of bone affected and the changes that occurred during the period of healing of the osteomyelitis.

In a case in which the operation was done by an orthopedic associate of mine, the roentgenograms showed clearly the healing of a severe osteomyelitis of the scapula. In this case, the operative procedure was simply that of drainage, without any curetting, and no attempt was made at any time during the convalescence to remove the diseased bone.

THE SO-CALLED BURSA PHARYNGEA IN MAN

ITS ORIGIN, RELATIONSHIP WITH THE ADJOINING NASOPHARYNGEAL STRUCTURES AND PATHOLOGY *

GEORGE MORRIS DORRANCE, M.D.

Professor of Maxillo-Facial Surgery, the Thomas W. Evans Museum and Dental Institute School of Dentistry, University of Pennsylvania; Surgeon to St. Agnes Hospital and the American Oncologic Hospital

PHILADELPHIA

I became interested in the bursa pharyngea after I had seen it in an adult with a cleft palate. My patient presented a condition seldom seen with the rhinoscope, as figure 1 shows. In this case there was no tonsillar adenoid tissue in the pharynx, and yet there was a distinct sac or pouch, the opening of which was situated above the upper border of the superior constrictor muscle of the pharynx. This opening could be probed for perhaps half an inch.

A picture of the position of the bursa is shown in figure 2, which is taken from Toldt's [1] "Atlas of Human Anatomy." Figure 3 shows the bursa in a case of cleft palate reported by Carwardine,[2] and here is seen its relationship with the pharyngeal tonsil.

Just what is this bursa pharyngea, and has it any relationship with cleft palate? The term bursa identifies a closed sac lined with endothelium. While the so-called bursa pharyngea is not a closed sac nor of mesodermic origin, long usage justifies the application of the term. The term bursa pharyngea designates a saclike depression in the posterior wall of the nasopharynx just above the uppermost fibers of the superior constrictor muscle, and usually extending upward and backward toward the occipital bone which it sometimes reaches with its apex (fig. 4). It is a well marked structure in the human embryo, although it seems to be rare as a distinct structure in the adult.

I consulted the standard textbooks on embryology and rhinology and was surprised to find that there was considerable confusion con-

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* Submitted for publication, May 23, 1930.

* The author was assisted in the preparation of this article by Dr. Milton K. Meyers and Dr. Enayat Shirazy.

1. Toldt, Carl: An Atlas of Human Anatomy for Students and Practitioners, translated by M. Eden Paul, New York, The Macmillan Company, 1926, vol. 2, fig. 710, p. 436.

2. Carwardine, Thomas: The Morphology and Pathology of the Pharyngeal Pouch, Bristol Med.-Chir. J. **15**:310, 1897.

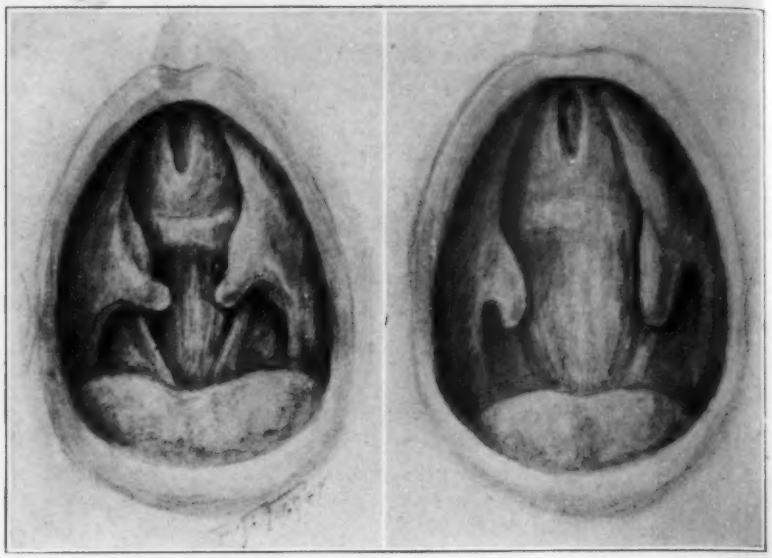

Fig. 1.—Artist's presentation of the bursa pharyngea in an adult patient with cleft palate. Note Passavant's cushion formed by the pterygopharyngeus portion of the superior constrictor muscle of the pharynx as the patient makes an effort at gagging. The sides of the cleft in the velum tend to approximate toward the mesial line; the tips of the cleft uvula are rotated inward.

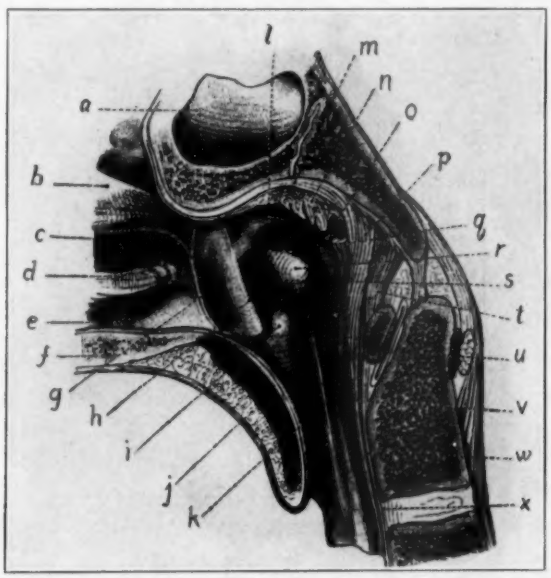

Fig. 2.—Median section of the human pharynx (from Toldt's Atlas of Human Anatomy) shows the position of the bursa pharyngea and the adjoining pharyngeal tissue. Note the relation of the bursa to the pharyngeal tonsil. *a* indicates sphenoidal sinus; *b*, middle turbinate bone of the nose; *c*, middle meatus of the nose, *d*, inferior turbinate bone of the nose; *e*, inferior meatus of the nose; *f*, hard palate; *g*, nasopharyngeal part of the inferior meatus, or posterior nasal sulcus; *h*, nasopharyngeal or salpingopalatine fold; *i*, levator cushion; *j*, soft palate, or velum pendulum palati; *k*, lateral wall of the pharynx; *l*, arched summit of the pharynx; *m*, fibrocartilage of the foramen lacerum medium; *n*, pharyngeal tonsil; *o*, pharyngeal bursa; *p*, pharyngeal aponeurosis; *q*, lateral recess of the pharynx, or fossa of Rosenmüller; *r*, cushion of the eustachian orifice; *s*, pharyngeal orifice of the eustachian tube; *t*, anterior occipito-atlantal ligament; *u*, salpingopharyngeal fold; *v*, posterior wall of the pharynx; *w*, prevertebral fascia, and *x*, posterior pillar of the fauces.

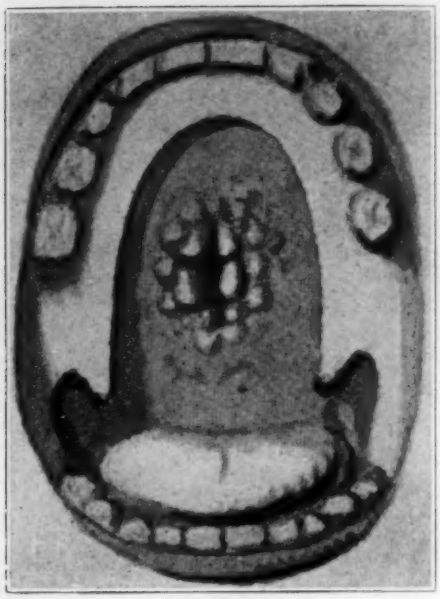

Fig. 3.—The bursa pharyngea in a case reported by Carwardine (*Bristol Med.-Chir. J.* **15**:310, 1897). Here is seen its relationship with the pharyngeal tonsil.

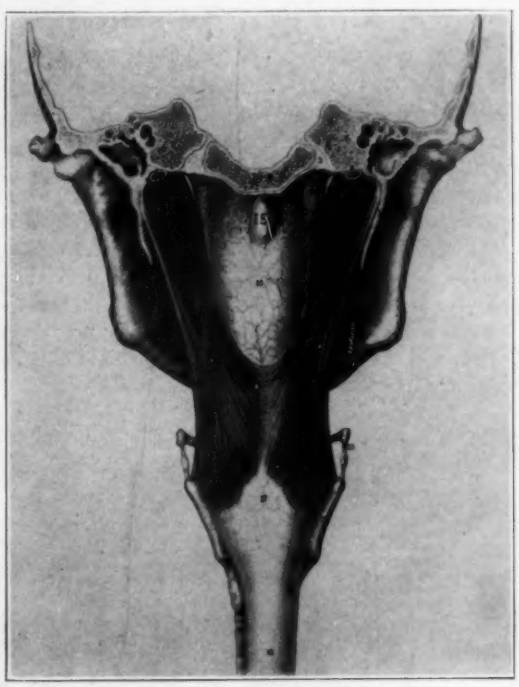

Fig. 4.—Dorsal view of the pharynx: *15,* the bursa pharyngea. Note its relationship to the occipital bone (from von Luschka: Der Schlundkopf des Menschen).

cerning its origin, and that the investigator cannot get a definite idea as to this structure.

Thus Heisler,[3] in his textbook on embryology, stated that the bursa represents the craniopharyngeal canal and identified it with Rathke's pouch. Sir St. Clair Thomson,[4] the eminent rhinologist, expressed the same opinion. Many clinicians regard it as Rathke's pouch or as the median recess of the pharyngeal tonsil or do not give its origin much thought.

Perhaps some of the prevailing confusion in the minds of the medical profession concerning the origin and significance of this structure is due to the lack of clearness of the views with respect to the bursa pharyngea.

In order to present to the medical profession, with some degree of clearness, the origin and significance of the bursa pharyngea, I have made the following survey of the literature.

HISTORICAL SURVEY

In 1842, Mayer[5] of Bonn, in an article from which a free translation is here submitted, first described in man an invagination of the pharynx that is supposed to have been the structure subsequently called bursa pharyngea:

> In April of 1840, I described a bursa in several vertebrates and made the statement that only a rudiment of it was found in man. I have since found this bursa in several human cadavers. The bursa lies as a sac in the mucosa covering the body of the sphenoid bone either behind the choanae or else just behind the alae of the vomer. The opening of this bursa varies in size, the largest could hold a cherry-pit. A septum may divide the opening to it. I am as yet unable to say whether or not the sac is in connection with the sphenoid sinuses. However, the following facts would lead me to suspect this: In almost all mammals there is an opening in the sella turcica which leads into the sinus of the body of the sphenoid bone. In addition, there is another opening at the base of the skull leading directly from the sphenoid sinus where the bursa pharyngea is found. In man, such openings in the sella turcica and at the base of the skull are rarely found. They may be so small as to be taken for nutrient foramina.

The bursa which Mayer,[5] in 1842, described in man was not a structure exactly similar to those which he saw and which he described

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3. Heisler, J. C.: Text-Book of Embryology for Students of Medicine, ed. 3, Philadelphia, W. B. Saunders & Company, 1907.

4. Thomson, St. C.: Diseases of the Nose and Throat, ed. 3, London, Cassell & Company, Ltd., 1926.

5. Mayer, F. J. C.: Neue Untersuchungen aus dem Gebiete der Anatomie und Physiologie, Bonn, 1842; quoted by Bloch: Ueber die Bursa pharyngea, Berl. klin. Wchnschr. **25**:269, 1888.

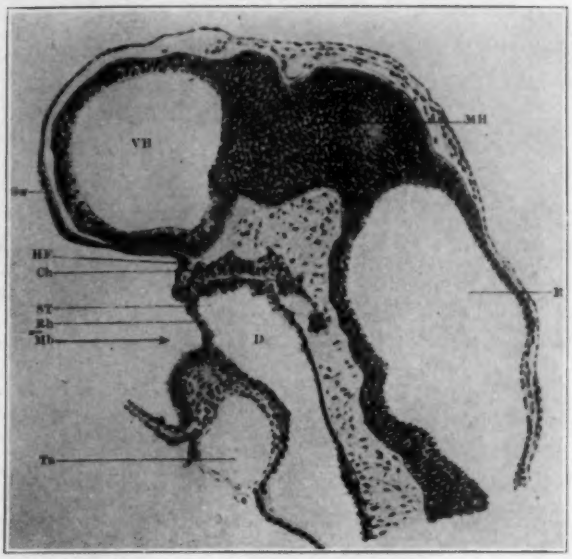

Fig. 5.—The head end of a rabbit embryo (from Fischel: Lehrbuch der Entwicklung des Menschen, Berlin, Julius Springer, 1929): *HF*, hypophyseal groove *Ch*, notochord; *ST*, Seessel's pocket; *Rh*, basal membrane; *Ta*, truncus arteriosus; *Mb*, stomodeum; *D*, primitive pharynx; *R*, rhombencephalon, and *VH*, forebrain.

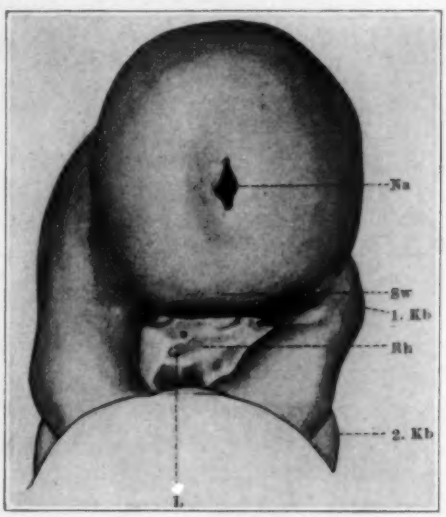

Fig. 6.—Head end of a human embryo with eighteen somites showing openings in the buccopharyngeal membrane (from Fischel, Sternberg's model): *Na*, anterior neuropore; *Rh*, basal membrane; *L*, opening in the buccopharyngeal membrane; *1.Kb*, first brachial arch; *2.Kb*, second brachial arch, and *Sw*, frontal cushion.

in his article of 1840.[6] In the earlier publication, he described a pouch in association with each of the eustachian tubes. These pouches met in the median line of the pharynx. He spoke here of the structure in man as a small groove and as situated laterally in the upper portion of the posterior wall of the pharynx.

It is possible that in his later communication, that of 1842, Mayer confused the true pharyngeal bursa with the pouch of Rathke, a structure that had been described in detail a few years previously by Rathke. Rathke[7] described his pouch as the embryonic pharyngeal opening of the craniopharyngeal canal, which leads to the base of the brain, trans-

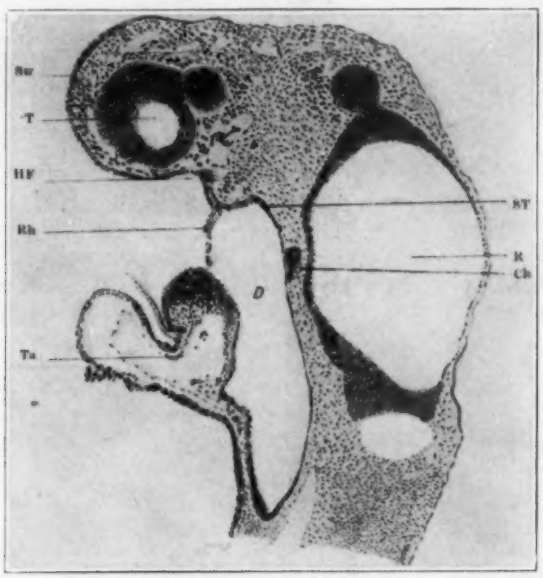

Fig. 7.—Head end of a rabbit embryo showing the beginning of rupture of the buccopharyngeal membrane *Rh* (from Fischel). Note position of the notochord at *Ch*. *T* indicates the telencephalon.

mitting an invagination that goes to form the anterior lobe of the hypophysis. Subsequent study has shown that remnants of this invagination persist in the lining of the posterior wall of the pharynx as the pharyngeal hypophysis. The duct with its pharyngeal opening, Rathke's pouch, is normally present in early embryonal life in man, being derived, in its early form at least, solely from the ectoderm of the primitive

6. Mayer, F. J. C.: Bursa seu Cystis tubae eustachianae bei einige Säugetieren, Neue Notizen aus dem Gebiete der Natur- und Heilkunde **14**:1, 1840.

7. Rathke, H.: Ueber die Entstehung der Glandula pituitaria, Arch. f. Anat., Physiol. u. wissensch. Med., 1838, p. 482.

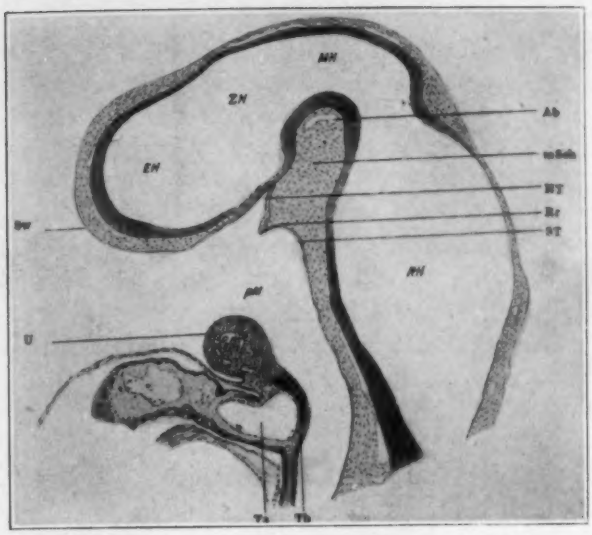

Fig. 8.—Head end of a rabbit embryo at a stage of development later than that shown in figure 7 (from Fischel): *HT*, Rathke's pouch; *Rr*, remains of buccopharyngeal membrane; *ST*, Seessel's pocket; *U*, mandibular process; *pM*, primary mouth cavity; *RH*, rhombencephalon; *MH*, midbrain; *ZH*, 'tween-brain, and *EH*, end-brain.

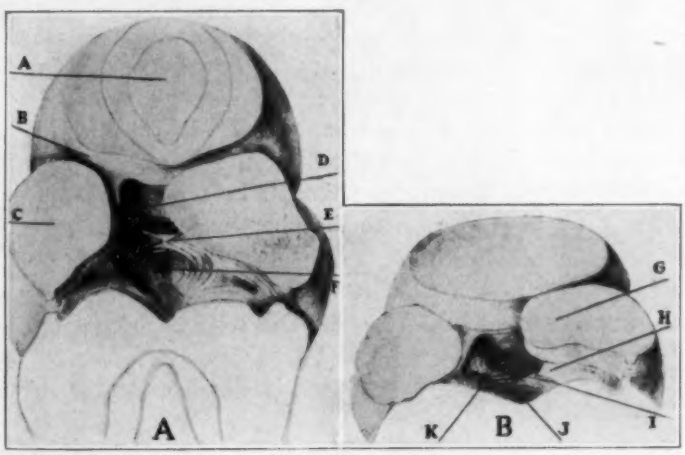

Fig. 9.—Head-on pictures of appearance of the pharynx of pig embryo after rupture of the buccopharyngeal membrane (from Heller and Mori: Ueber die Bildung der Hypophyse bei Säugetieren, *Ztschr. f. d. ges. Anat.* **76**:165, 1925); *a*, forebrain; *b*, jaw-eye cleft; *c*, maxillary arch cut in median plane; *d*, hypophyseal anlage; *e*, remains of buccopharyngeal membrane; *f*, Seessel's pocket; *g*, *h*, *i*, remains of buccopharyngeal membrane; *j*, Seessel's pocket, and *k*, hypophyseal anlage.

mouth cavity or stomodeum. Immediately caudad to Rathke's pouch, in the early embryo, is the buccopharyngeal membrane which is usually described as made of a layer of ectoderm and another of entoderm. This membrane later ruptures. In the embryo, immediately caudad to the buccopharyngeal membrane, is a short invagination known as Seessel's pocket.

The relations of Rathke's pouch and Seessel's pocket to the buccopharyngeal membrane are well shown in figures 5, 6, 7, 8 and 9.

Rathke's pouch is an embryonic structure which rarely remains patulous in adult life. Figure 10, from Suchannek,[8] shows such a persistence. Figure 11 *A* and *B*, from Testut,[9] shows the structures that remain as vestiges of Rathke's invagination as well as the pharyngeal bursa.

Immediately cephalad to Seessel's pocket, a membranous fold becomes somewhat accentuated and, according to Minot,[10] is not exclusively the remains of the buccopharyngeal membrane. He regarded it as the homolog of the upper lip of the petromyzon or lamprey. This interpretation is not universally accepted.

Seessel [11] regarded the hypophyseal sac (with Rathke's pouch) as a derivative of the primitive gut. He spoke of the pocket named after him as an invagination in close juxtaposition with the hypophyseal sac, a structure which in the chick embryo becomes a branch of the hypophyseal cord. If this is true, entodermal tissue would seem to branch directly from the ectoderm of the cord or stalk, for subsequent to the publication of Seessel's article it was proved that the tissue of the hypophyseal cord is derived from the ectoderm. Seessel spoke also of an accessory pocket near the first one that he described. This second pocket, he believed, became a solid collection of cells, the fate of which he did not definitely determine, although he thought that these cells might have something to do with the pharyngeal tonsil.

Nusbaum [12] carefully studied the fate of Seessel's pocket in mammals. He obtained pictures (fig. 12 *A*, *B*, *C* and *D*) which, while resembling in certain respects those described by Seessel in chick embryos, show

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8. Suchannek: Ein Fall von Persistenz des Hypophysenganges, Anat. Anz. **2**:520, 1887.

9. Testut, L.: Traité d'anatomie humaine, ed. 7, Paris, O. Doin, 1923, vol. 4, p. 923.

10. Minot, C. S.: Human Embryology, ed. 2, New York, The Macmillan Company, 1897.

11. Seessel, A.: Zur Entwicklungsgeschichte Vorderdarms, Arch. f. Anat. u. Physiol., 1877, p. 449.

12. Nusbaum, J.: Einige neue Thatsachen zur Entwicklungsgeschichte der Hypophysis cerebri bei Säugetieren, Anat. Anz. **12**:161, 1896.

that the cord of cells that persists as the remains of the invagination represented by Seessel's pocket does not branch directly from the pharyngeal stalk.

At the present time, Seessel's pocket is held to be a structure that in the main pertains only to the embryo and that is of little or no importance.

The openings into the sella turcica mentioned by Mayer must be rare in man. Virchow [13] spoke of a nutrient foramen in the base of the sella turcica in man, and stated that he had never seen a perforation of any size in the posterior wall of the sphenoid sinus except as the result of disease. Zuckerkandl [14] said that he had seen communications between the sphenoid sinus and the middle fossa of the skull, but did not state specifically that these are pathologic.

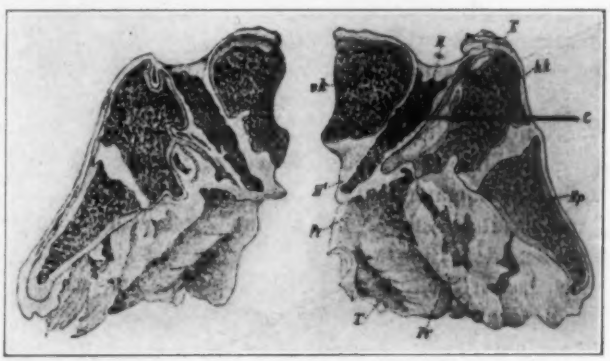

Fig. 10.—A rare case of persistence of the craniopharyngeal canal at a site anterior to where the bursa pharyngea would be (from Suchannek: *Anat. Anz.* **2**:520, 1887): *vk*, anterior portion of body of sphenoid bone; *hk*, posterior portion of body of sphenoid bone; *Hp*, body of occipital bone; *E*, exostosis; *T*, opening of eustachian tube; *C*, craniopharyngeal canal; *H*, upper portion of anterior lobe of hypophysis, region of sella turcica; *H'*, lower portion of anterior lobe of hypophysis, in pharyngeal membrane; *Pt*, upper edge of pharyngeal tonsil, and *Pt'*, lower edge of pharyngeal tonsil.

The craniopharyngeal canal sometimes runs close to the sinus, as figure 13 shows, and there seems to be no reason to suppose that a sphenoid sinus may not at times invade the course of a craniopharyngeal canal and thus produce a picture similar to those seen by Mayer in animals. The close proximity, in man, of a persistent craniopharyngeal canal to the sphenoidal sinus is shown in figure 14 from the sphenoidal bone of an adult rabbit. This figure shows how the craniopharyngeal

13. Virchow, R.: Untersuchungen über die Entwicklung des Schaedelgrundes, Berlin, Reimer, 1857, p. 42.

14. Zuckerkandl, E.: Normale und pathologische Anatomie der Nasenhöhle und ihrer pneumatischen Anhänge, Vienna, Wilhelm Braumüller, 1882, p. 173.

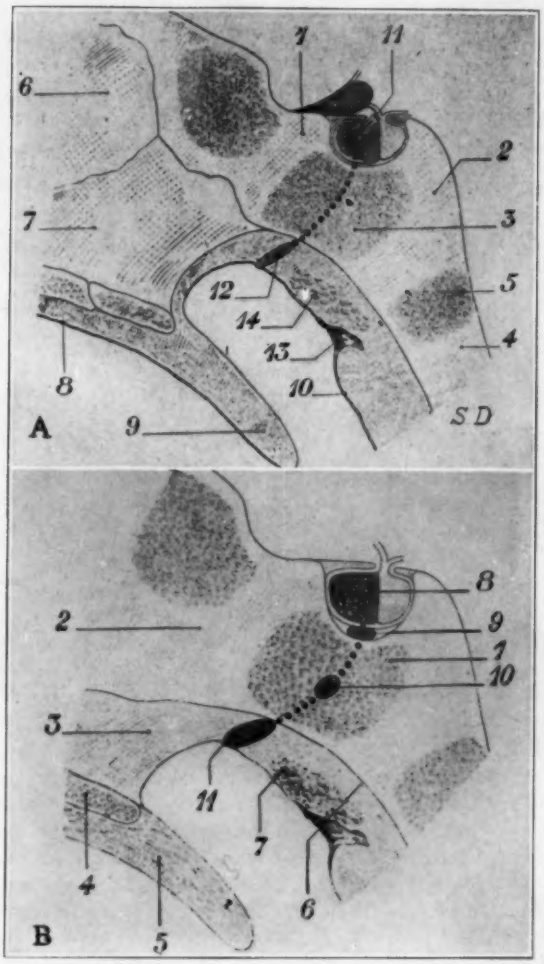

Fig. 11.—*A*, sagittal section to show the pharyngeal hypophysis in the newborn (from Testut: Traité d'anatomie humaine). Dotted line depicts the craniopharyngeal canal. *1*, suture between anterior and posterior portions of sphenoid bone; *2*, occipitosphenoid suture; *3*, ossified segment of posterior portion of sphenoid bone; *4*, basilar apophysis of occipital bone; *5*, a portion that is ossified; *6*, perpendicular plate of ethmoid bone; *7*, vomer; *8*, palatine vault; *9*, uvula; *10*, posterosuperior wall of pharynx; *11*, anterior lobe of cerebral hypophysis; *12*, pharyngeal hypophysis; *13*, bursa pharyngea, and *14*, pharyngeal tonsil. *B*, diagrammatic presentation of supernumerary hypophysis shown in *A* (from Testut). Dotted line shows course of craniopharyngeal canal. *1*, posterior part of sphenoid bone; *2*, anterior part of sphenoid bone; *3*, vomer; *5*, uvula; *6*, vault of pharynx; *7*, pharyngeal tonsil; *8*, cerebral hypophysis with two lobes; *9*, accessory intracranial hypophysis; *10*, accessory intra-osseous hypophysis, and *11*, pharyngeal hypophysis.

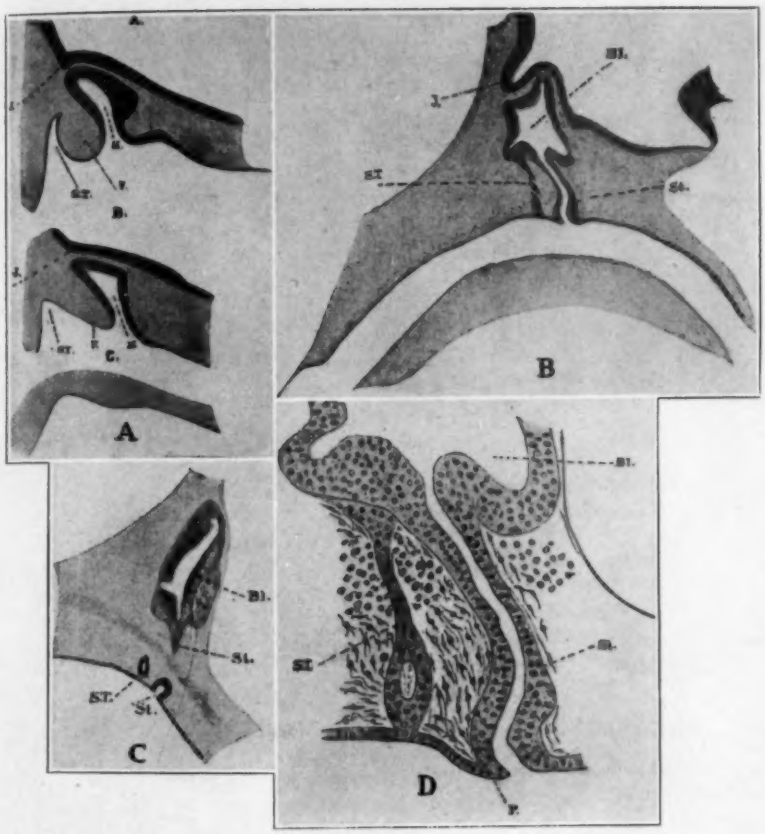

Fig. 12.—*A*, median sagittal section (A) and lateral sagittal sections (B and C) of dog embryo, 9 mm. long, through the region of Rathke's pouch and Seessel's pocket (from Nusbaum: *Anat. Anz.* **12**:161, 1896); H, Rathke's pouch; F, fold; ST, Seessel's pocket, and J, infundibulum; *B*, older dog embryo than that in *A*, showing the remains of Seessel's pocket and the hypophyseal stalk which is formed from Rathke's pouch (from Nusbaum); BL, hypophyseal cavity; st, remains of Rathke's pouch, and ST, remains of Seessel's pocket; *C*, section from dog embryo from 15 to 16 mm. long, showing remains of Seessel's pocket and the hypophyseal stalk formed from Rathke's pocket (from Nusbaum): *D*, sagittal section of the anlage of the hypophyseal stalk or duct of embryo, 14 mm. long (from Nusbaum); BL, hypophyseal sac; st, hypophyseal stalk or duct, and ST, remains.

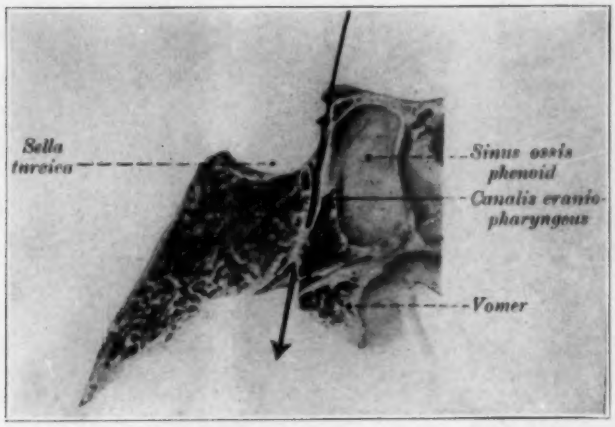

Fig. 13.—Arrow is shown within the craniopharyngeal canal which runs close to the sinus in this specimen (from Sokolow: *Arch. f. Anat. u. Physiol.*, 1904, p. 69).

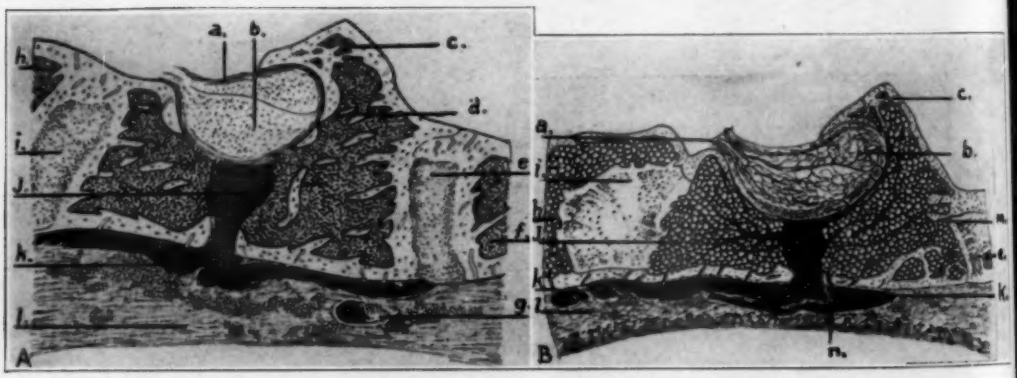

Fig. 14.—*A*, sagittal section through the middle of hypophysis, postsphenoid bone and nasal mucosa of adult rabbit; *B*, reconstruction from three sagittal sections through the middle of hypophysis, postsphenoid bone and pharyngeal mucosa of adult rabbit (both illustrations from Arai: *Anat. Heft.* **33**:413, 1907): *a.*, dura; *b.*, hypophysis; *c.*, dorsum sellae; *d.*, sphenoidale; *e.*, synchondrosis spheno-occipitalis; *f.*, basilare; *g.*, tuba auditiva; *h.*, presphenoid; *i.*, synchondrosis intersphenoid; *j.*, sinus venosus ossis sphenoidalis; *k.*, vena vertebralis mediana; *l.*, mucosa naris; *m.*, spongiosa postsphenoidalis, and *n.*, artery.

space is filled with a venous sinus, which in the rabbit corresponds to the location of the sphenoidal sinus. There are openings leading to and from this venous sinus connecting the space with the sella turcica and the base of the cranium, respectively (figs. 15, 16, 17, 18 and 19).

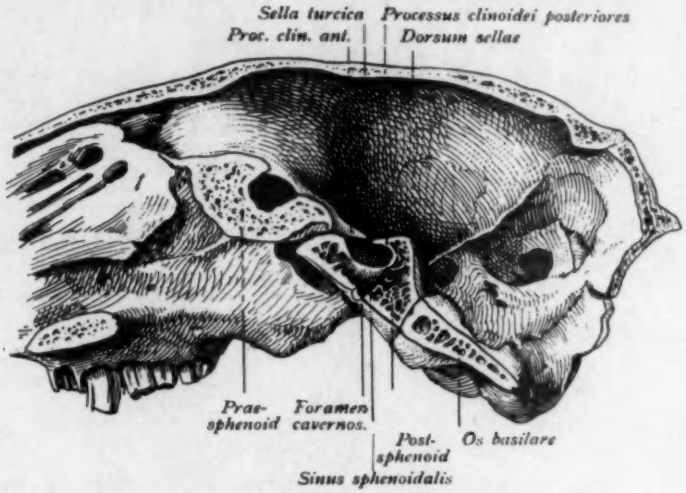

Fig. 15.—Cross-section through skull of a rabbit to show the craniopharyngeal canal, from Arai.

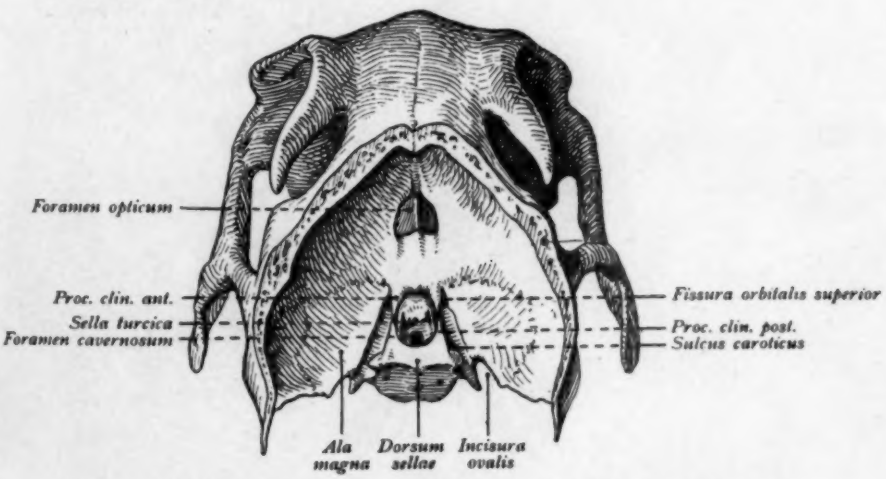

Fig. 16.—Anterior half of base of skull of rabbit embryo to show foramen cavernosum, the upper opening of craniopharyngeal canal (from Arai).

It is interesting to mention that Waldeyer,[15] from personal examination of complete collected material of 6,281 adult human skulls,

15. Waldeyer, W.: Bemerkungen über Gruben, Kanäle und einige andere Besonderheiten des Grundbeins (Os basilare), Internat. Monatschr. f. Anat. u. Physiol., 1905, vol. 21.

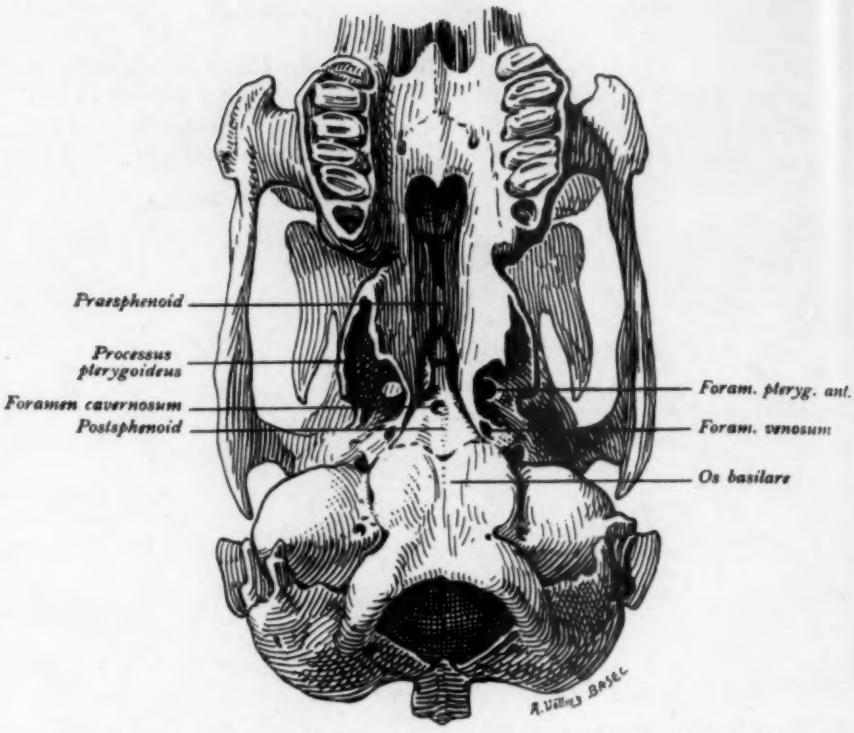

Fig. 17.—Base of skull of rabbit embryo seen from below to show the foramen cavernosum, the lower opening of craniopharyngeal canal (from Arai).

Fig. 18.—Base of skull seen from above to show the superior opening (indicated by dotted line) of the craniopharyngeal canal in man (from Sokolow: *Arch. f. Anat. u. Physiol.*, 1904, p. 69).

including his own material of 1,000 human skulls, found a persistent craniopharyngeal canal in only 0.25 per cent. He also found in 26 gorillas, 2 complete craniopharyngeal canals, 1 of which led into the sphenoidal sinuses.

The next author to describe the bursa pharyngea was Tourtual,[16] who called it Mayer's bursa pharyngea. Tourtual described it as a sac-like invagination of the pharyngeal mucosa having the average size of a pea. Its opening is oval, as a rule, and may be as small as the head

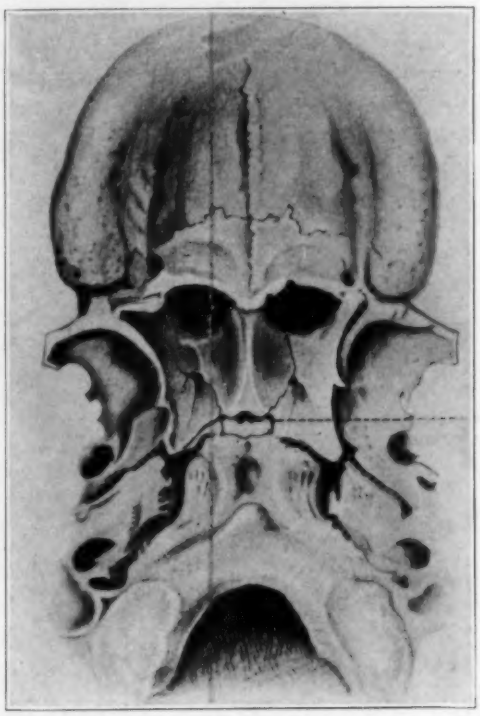

Fig. 19.—Base of skull seen from below to show the inferior opening (indicated by dotted line) of the craniopharyngeal canal of a different skull than that shown in figure 18 (from Sokolow).

of a pin or as wide as the width of the bursa itself. Tourtual did not concern himself with the embryology of the bursa, having confined his studies to its occurrence in adults. He did not accept the fact that it communicated by means of a canal with the cranial cavity. In certain cases, however, he found it embedded in a fovea bursa on the inferior aspect of the basilar process of the occipital bone.

16. Tourtual, C. T.: Neue Untersuchungen über den Bau des menschlichen Schlund- und Kehlkopfes, Leipzig, 1846; quoted by Bloch: Berl. klin. Wchnschr. **25**:269, 1888.

I may remark here parenthetically that this fovea bursa, which is shown in figure 20, has been studied thoroughly by Tourneux [17] who called it the fossette pharyngienne. He stated that Romiti [18] regarded it as an excavation to accommodate Seessel's pocket. The fovea bursa had been considered by Perna [19] as the site where the notocord comes into direct contact with the perichondrium on the ventral aspect of the basioccipital bone. Tourneux assumed that this structure corresponds with the base of the bursa pharyngea, and he distinguished it from the fossette naviculaire, a shallower depression in which it is often located. Poelchen,[20] before Tourneux, made a similar distinction, using the terms

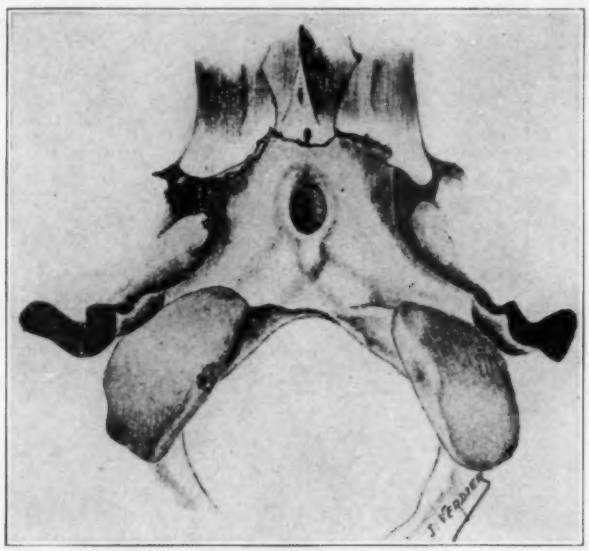

Fig. 20.—The two concentric depressions on the basilar process of the occipital bone. The innermost of these depressions, the fossa pharyngienne represents the place of contact of the bursa pharyngea. The outermost, the fossa naviculaire, a shallower depression, represents what is supposed to be the indentation produced by the notochord. Modified from Tourneux (*J. de l'anat. et de la physiol.* **48**:516, 1912) after a specimen from the anatomic museum of Toulouse.

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17. Tourneux, J. P.: Bourse pharyngienne et récessus median du pharynx chez l'homme et chez le chéval, fossettes pharyngiennes et naviculaires chez l'homme, J. de l'anat. et de la physiol. **48**:516, 1912.

18. Romiti, G.: Fosetta faringea dell'osso occipitale nell'uomo, Atti d. Soc. Toscana di sc. nat., Pisa, 1889; Tourneux (footnote 17); La fosetta faringea dell' osso occipitale nell'uomo, Atti d. Soc. Toscana di sc. nat., Pisa, 1890; quoted by Tourneux (footnote 17).

19. Perna, G.: Sul canale basilare e sul significato della fossette faringea dell' osso occipitale, Anat. Anz. **28**:379, 1906.

20. Poelchen, R.: Zur Anatomie des Nasenrachenraumes, Virchows Arch. f. path. Anat. **119**:118, 1890.

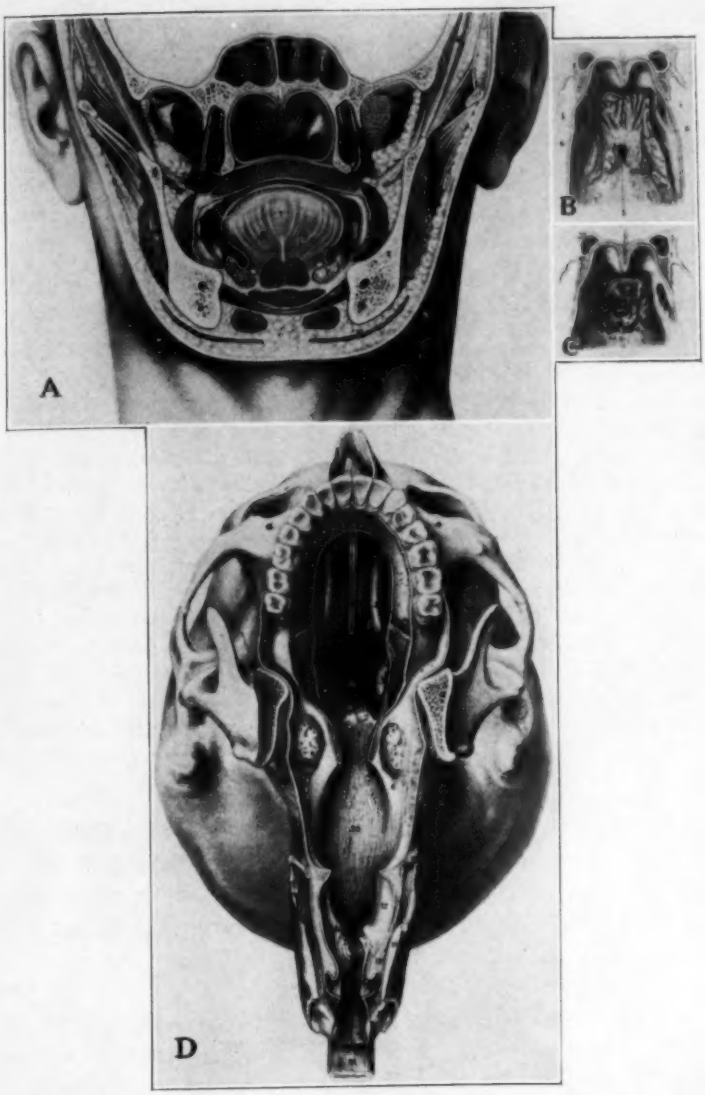

Fig. 21.—Four views of the pharynx to show the bursa pharyngea (reproduced from von Luschka: Der Schlundkopf des Menschen): *A*, coronal section of the head to show at *4* the entrance into the bursa pharyngea; *B*, the opening into the bursa pharyngea at *5; C*, the opening into the bursa pharyngea at *5;* at *7* is shown one of the tonsillar spaces; *D*, ventral view of the pharynx to show at *19* the bursa pharyngea.

foramen caecum and fossa navicularis, respectively, for the fossette pharyngienne and the fossette naviculaire. Tourneux interpreted the occurrence of these two concentric depressions in the following words: "Both depressions are the result of indentations made by the notochord and by the bursa pharyngea, but the latter may make a deeper impression, thus forming the fossette pharyngienne." It is of interest to add here that Waldeyer found no fovea bursa in the anthropoid apes, since these apes lack the bursa pharyngea.

Von Luschka,[21] in his work on the pharynx, described the bursa pharyngea and depicted it in several illustrations (fig. 21). He was not acquainted with its embryology and erroneously brought it in developmental relationship with the hypophysis. His description, which is translated freely, is submitted:

> This opening is the opening into an oblong pouch-like appendage of the vault of the pharynx at most 1.5 cm. long and 6 mm. broad, which is blended with the adenoid tissue in front of it by means of a loose cellular stratum. This appendage extends to the body of the occipital bone to become imbedded into the external fibrous envelope of this segment of bone by means of its attenuated and sometimes sharply pointed extremity. Posteriorly, the bursa is usually in direct contact with acinous glands, and sometimes to the side of it is found a muscle which has its origin by a flat tendon from the fibrous tissue of the body of the occipital bone. (This muscle is evidently the longus capitis. Figure 22, from Killian,[22] shows the relationship of the bursa to the muscle very well.) The bursa is covered behind by the beginning of the fibrous ligamentum pharyngis medium which, as is known, has its origin from the tuberculum pharyngeum. . . . The wall of the bursa consists chiefly of adenoid tissue and is from 0.5 to 1.5 mm. in thickness. It has a mucous surface which is not as a rule smooth, the mucosa being provided with irregular rugged processes and sometimes being disposed in longitudinal folds. At times the narrowed upper end closes off, thus leading to the formation of a cyst.

Figure 23 shows an illustration of a cyst of the retropharynx, probably a cyst of the bursa pharyngea in an 8 months fetus. Figure 24 shows another cyst of the bursa, surrounded with adenoid tissue. Figure 25 *A* and *B* depicts another bursa pharyngea, together with the microscopic section of its wall.

Dursy [23] was the first to make a comprehensive study of the development of the bursa pharyngea. His observations did not reveal any pocket-like structure in the human pharynx which could be brought into relation with openings at the base of the skull. Dursy found the bursa pharyngea situated under the basilar process of the occipital

21. von Luschka, H.: Der Schlundkopf des Menschen, Tübingen, Laupp, 1868.

22. Killian, G.: Ueber die Bursa und Tonsilla pharyngea, Morphol. Jahrb. **14**:618, 1888.

23. Dursy, E.: Zur Entwicklungsgeschichte des Kopfes des Menschen und höheren Wirbeltiere; quoted by Bloch: Berl. klin. Wchnschr. **25**:269, 1880.

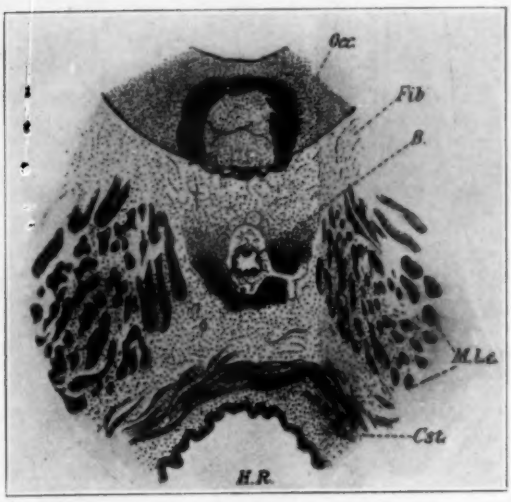

Fig. 22.—Transverse section of the dorsum of the pharynx of 21 cm. embryo to show the bursa pharyngea lying between the fibers of the longus capitis muscles (from Killian: *Morphol. Jahrb.* **14**:618, 1888): *M.l.c.*, longus capitis muscle; *Cst.*, superior constrictor muscle of the pharynx; *B.*, "bursa pharyngea"; *Fib.*, fibrocartilage, and *Occ.*, occipital bone.

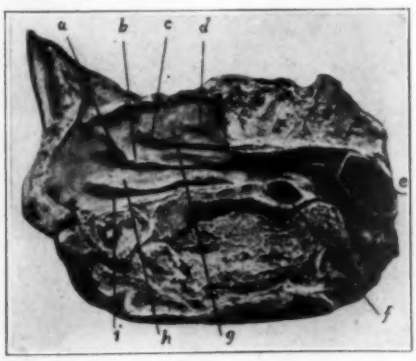

Fig. 23.—A sagittal section of an 8 months fetus, natural size, showing a cyst of the retropharynx and cyst of the palate. The cyst in the pharynx is probably one of the bursa pharyngea (from Orondi: Handbuch der speziellen Chirurgie des Ohres und der oberen Ludtwege, Würzburg, 1912, vol. 1, plate 50, fig. 29).

bone without any relationship with Rathke's pouch. In all the older embryos that he studied, the mucous membrane was intimately adherent to the occipital bone at the site of junction of the roof of the pharynx with the posterior pharyngeal wall, forming at this junction a funnel-shaped depression. This depression persists and deepens into a bursa pharyngea by the growth of the surrounding mucous membrane.

Ganghofner [24] claimed that a bursa proper forms only as a result of adhesions between portions of the pharyngeal tonsil, a condition that he regarded as always pathologic in adults. He also denied the penetration of the bursa into the occipital bone. He called the structure "recessus medius pharyngis," and regarded the recess as a part of the

Fig. 24.—Bursa pharyngea surrounded by adenoid tissue, one-half natural size (from Meyer: *J. Anat. & Physiol.* **38**:107, 1913-1914).

median split of the tonsil. The apex of the bursa may be fixed, however, in embryonal life and the bursa may elongate by the forward growth of tonsillar tissue around it.

Froriep,[25] in 1882, offered an explanation that appears in harmony with modern research on the origin of the bursa pharyngea. He saw the bursa in an embryo that measured 3.8 cm. in length. In this specimen the apex of the bursa was embedded in a collection of cellular

24. Ganghofner, F.: Ueber die Tonsilla und Bursa pharyngea, Sitzungsb. d. math. Naturw. Kl. d. kaiserl. Akad. d. Wissenschaft. **78**:182, 1879; quoted by Bloch: Berl. klin. Wchnschr. **25**:269, 1880.

25. Froriep, A.: Kopftheil der Chorda dorsalis bei menschlichen Embryonen, Festgabe für Jacob Henle, Bonn, 1882; quoted by Huber: Anat. Rec. **6**:373, 1912.

strands that belonged to the retropharyngeal notochord. He thought it possible that the site where the notochord joins the primitive retropharyngeal wall forms a point of attachment at which the notochord and the wall remain in apposition even after the rest of the wall of the pharynx has separated from the base of the skull. The bursa pharyngea, according to Froriep, is formed at the site of this attachment. An original illustration from Froriep is shown in figure 26.

Three years later, Tornwaldt [26] drew the attention of clinicians to the bursa, in a publication in which he held that diseases of the bursa pharyngea were responsible for many systemic disturbances. Perhaps

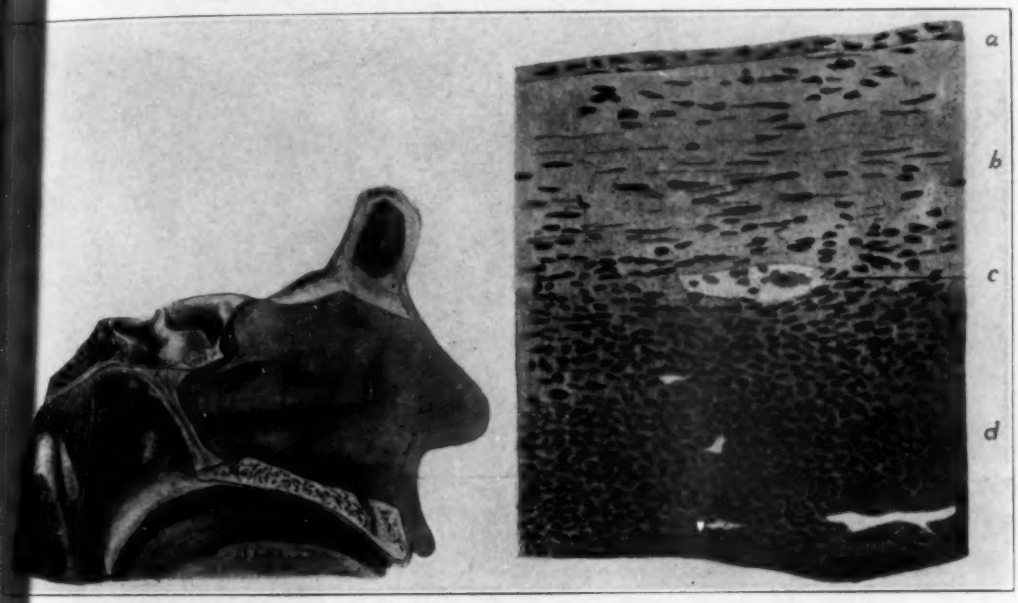

Fig. 25.—Left, bursa pharyngea, one-half natural size. Right, wall of bursa pharyngea: *a*, inner syncytial (?) layer; *b*, fibrous connective tissue; *c*, blood vessel, and *d*, adenoid tissue (from Meyer).

many of the bursae that Tornwaldt dealt with were merely central pocketings of the pharyngeal tonsil. On the other hand, he may have seen true bursae.

In the following year, Trautmann [27] called attention to a deepened furrow embedded between the ridges of the pharyngeal tonsil. This

26. Tornwaldt: Ueber die Bedeutung der Bursa pharyngea für die Erkennung und Behandlung gewisser Nasenrachenkrankheiten, Wiesbaden, J. F. Bergmann, 1885.

27. Trautmann: Anatomische, pathologische und klinische Studien über die hypertrophie der Rachentonsille, Berlin, 1886; quoted by Schwabach (footnotes 28 and 29).

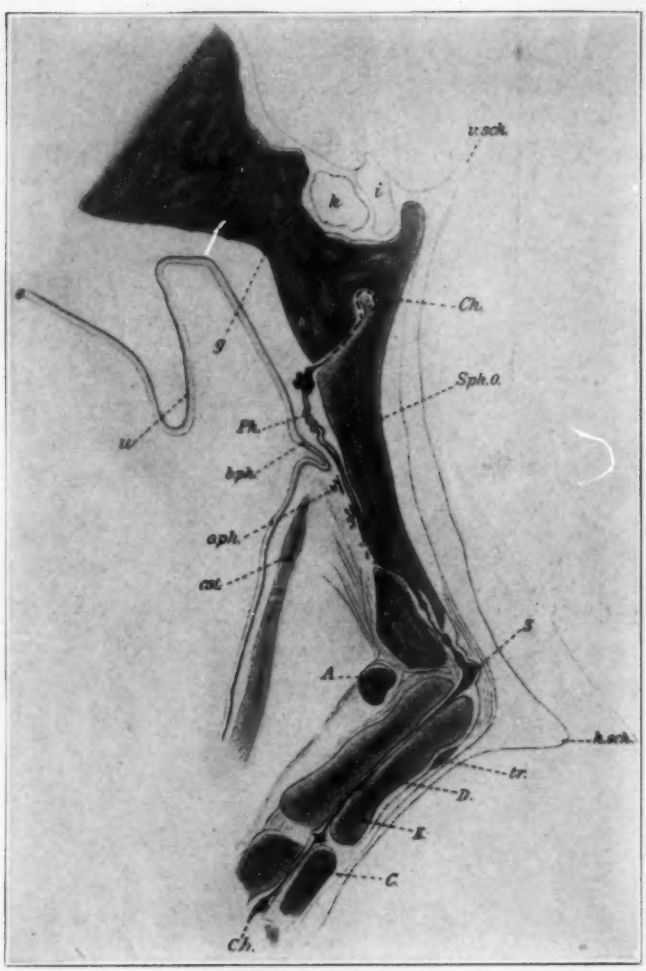

Fig. 26.—Reproduction of a section of Froriep's embryo (from Gradenigo: Die Hypertrophie der Rachentonsille) to show the close relationship of the notocord with the pharyngeal mucosa at the site of the bursa pharyngea: *Ch*, notocord; *b.ph.*, bursa pharyngea; *o.ph.*, ligamentum occipitopharyngeum; *Ph.*, roof of pharynx; *g*, craniopharyngeal canal or the hypophyseal duct; *u*, uvula; *Sph.O.*, spheno-occipital cartilage; *A*, arch of atlas; *B*, odontoid process of axis, and *E*, axis.

furrow, he asserted, sinks somewhat into the fibrocartilaginous tissue of the base of the skull without the formation of a canal or without entering the bone.

Schwabach [28] (1887), a supporter of Ganghofner's view, studied the pharyngeal tonsil in children, adults and a 6 months fetus. His observations did not reveal a bursa with walls as previously described by von Luschka. However, Schwabach saw in some cases a simple invagination in the posterior end of the median mucosal furrow of the tonsil (fig. 27). This invagination, he asserted, may be regarded in some cases as the site of union of the median tonsillar furrow with various lateral furrows which converge toward that in the median line. The depression was well marked when there was a median tonsillar ridge instead of a median groove; figure 28 shows a specimen with such a median ridge. In some cases, however, the posterior end of this median

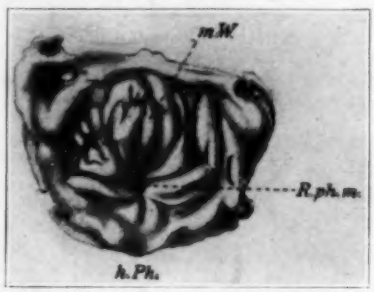

Fig. 27.—The pharyngeal tonsil of a child, aged 4 months (Schwabach's case after Gradenigo): *m.W.*, unpaired median cushion; *R.ph.m.*, median recess of the tonsil, and *h.Ph.*, posterior wall of the pharynx.

depression was completely isolated from the rest of the furrow; in other cases it was divided by a transverse fold into an anterior and a posterior depression (fig. 29).

In a later communication dealing with studies of the pharyngeal tonsil, Schwabach,[29] in 1888, made further investigations concerning the bursa pharyngea, using forty-five embryos. In embryos measuring less than 6 cm. in crown-rump length he found no evidence of any depression or infolding in the pharyngeal vault. In embryos of from 6 to 7 cm. in crown-rump length, he observed a shallow depression or groove in the midline at the transition of the fornix to the posterior wall which he regarded as the anlage of the pharyngeal tonsil. The

28. Schwabach, L.: Ueber die Bursa pharyngea, Arch. f. mikr. Anat. **29**:61, 1887.

29. Schwabach, L.: Zur Entwicklung der Rachentonsille, Arch. f. mikr. Anat. **32**:187, 1888.

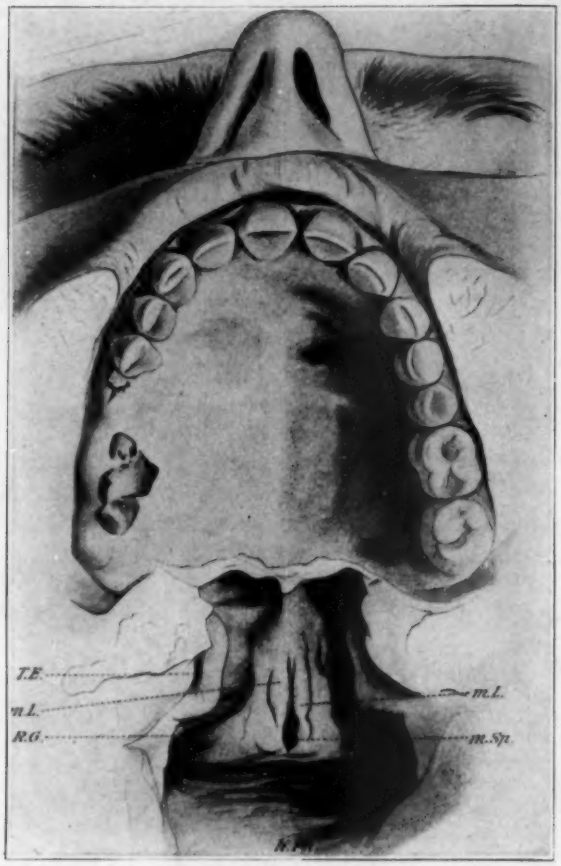

Fig. 28.—Ventral view of the pharynx of a woman, aged 40 (from Schwabach: *Arch. f. mikr. Anat.* **32**:187, 1888); *m.L.*, median ridge; *m.sp.*, median split; *T.E.*, orifice of eustachian tube; *R.G.*, fossa of Rosenmueller, and *h.Ph.*, posterior wall of the pharynx.

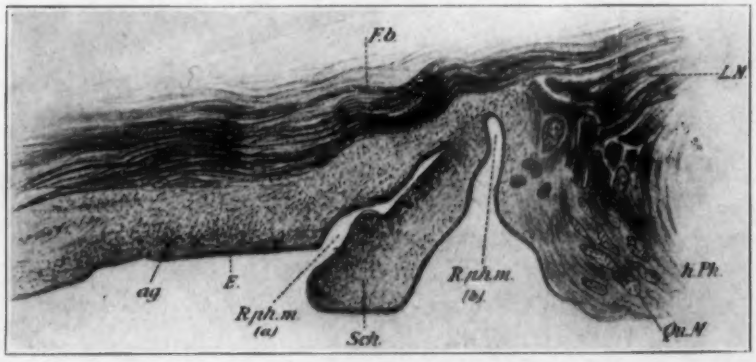

Fig. 29.—Median section of the pharyngeal tonsil of a new-born infant, showing division of the median recess into an anterior (*R.ph. m.* [*a*]) and a posterior (*R.ph.m.* [*b*]) part by means of a transverse fold of mucous membrane (*Sch.*) (from Schwabach, after Gradenigo): *ag.*, adenoid tissue; *E.*, epithelium; *h.Ph.*, posterior wall of the pharynx; *Qu.M.*, muscles transversely cut; *L.M.*, muscles longitudinally cut, and *F.b.*, basilar fibrocartilage.

depression reached its maximum depth in embryos of from 9 to 10 cm. in crown-rump length, becoming shallower in longer embryos as the development of the adenoid tissue of the pharyngeal tonsil proceeded. He believed that the invagination of the pharyngeal mucosa at the site of the bursa may be produced by the traction exerted by a compact band of connective tissue, previously described by Froriep and called by him the ligamentum occipito-pharyngeum. Figure 30 shows this ligament and its relationship to the bursa.

Killian studied the development of the pharyngeal tonsil and the bursa pharyngea. His researches (table) included as material sixty-five human embryos of from 3 to 8 months, three new-born infants and two older children. In twenty-eight, or 40 per cent, of the seventy cases

The Incidence of the Bursa Pharyngea in the Embryo or Very Young Child (According to Killian)*

Human embryos, 3 to 8 months	65	
New-born children	3	
Older children	2	
Total	70	
No evidence of the bursa pharyngea	28	(40%)
Well developed bursa pharyngea	14	(20%)
3 of these 14 were microscopic		
5 of these 14 were macroscopic		
Grooves or funnel-shaped depressions only	28	(40%)
9 of these 28 were microscopic		
19 of these 28 were macroscopic		
10 of these 28 showed evidence of the presence of the pharyngeal tonsil		

* Killian: Morphol. Jahrb. **14**: 618, 1888.

there was no evidence of a bursa; fourteen of the remaining cases presented a well developed bursa; the remaining thirty-eight cases showed more or less pronounced grooves or funnel-shaped depressions. This investigator criticized Froriep, who based his observations on a single case. Killian asked why the bursa should occur at a definite site, when there is more than one locality where the notochord and the pharyngeal mucous membrane come in contact in man. He also discussed the question of the influence of the ligamentum occipitopharyngeum on the formation of the bursa and concluded that the bursa was due to a "true epithelial outgrowth" rather than to "mechanical factors." Figure 31 from Killian shows the bursa of an embryo. Figure 32 from Killian shows that the bodies of the longus capitis do not influence the form of the bursa. Killian expressed the belief that the bursa invaginates independently of mechanical factors.

Killian insisted that the only mammal known to have a bursa pharyngea homologous to that of man was the marmot (*Arctomys*) (fig. 33), and that the pharyngeal pouches of the pig are not true bursae

pharyngeae in the sense that the human bursa is. Von Luschka had already pointed this out. It is true that in the pig the bursa lies much caudad to its relative position in man. One of these pouches in the pig is shown in figure 34. According to Fleming's translation and edition of Chauveau and Arloing's [30] work, "In the pig, the posterior part of the pharynx is narrow, and has a pouch immediately above the glottis,

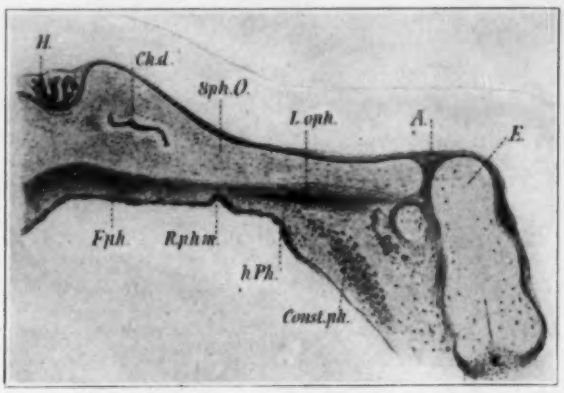

Fig. 30.—The "ligamentum occipito-pharyngeum" and its relationship to an indentation at the site of the bursa pharyngea (from Schwabach): *H.*, hypophysis cerebri; *Ch.d.*, notochord; *Sph.O.*, spheno-occipital cartilage; *L.oph.*, ligamentum occipito-pharyngeum; *Fph.*, fornix of the pharynx; *R.phm.*, median recess of the pharynx; *h.Ph.*, posterior wall of the pharynx, and *Const.ph.*, superior constrictor muscle of the pharynx.

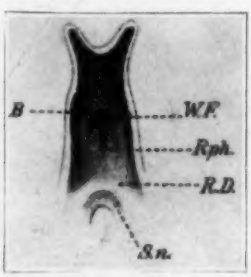

Fig. 31.—From the original of Killian (*Morphol. Jahrb.* **14**:618, 1888) to show the bursa pharyngea in a human embryo 19 cm. long; × 2. *B.*, opening of the bursa pharyngea; *Rph.*, raphe; *W.F.*, angle of fold; *R.D.*, roof of naso-pharynx, and *S.n.*, nasal septum.

between the thyro- and crico-pharyngeal muscles." According to Tourneaux, a bursa pharyngea homologous to that of man is found in the horse.

30. Chauveau, Auguste; and Arloing, S.: The Comparative Anatomy of the Domestic Animals, translated by George Fleming, London, J. & A. Churchill, 1873.

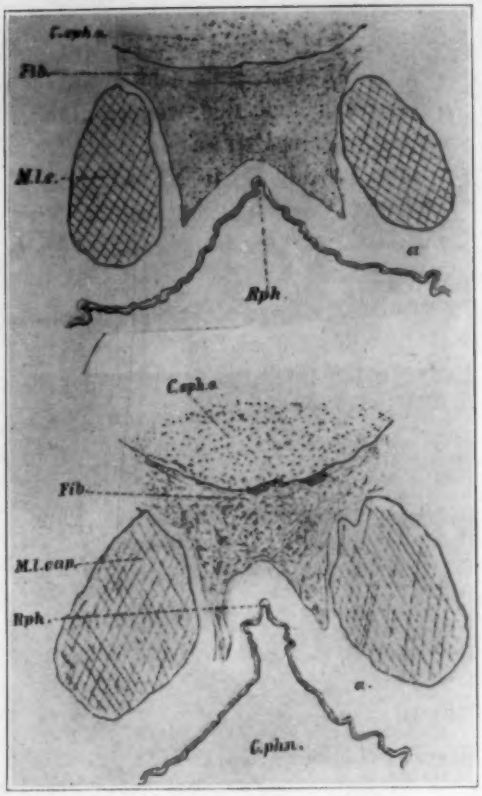

Fig. 32.—Frontal sections of a human embryo (from Killian) showing that muscle tissue does not influence the form of the bursa pharyngea, but that it seems to invaginate: *C.sph.o.*, spheno-occipital cartilage; *Fib.*, fibrocartilage; *M.l.cap.*, longus capitis muscle; *M.l.c.*, longus capitis muscle; *Rph.*, raphe, and *C.ph.n.*, nasopharynx.

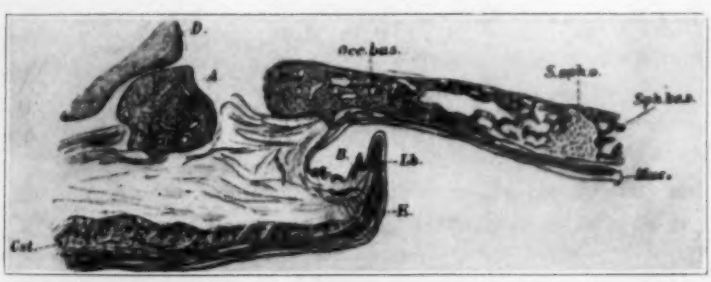

Fig. 33.—Sagittal section of posterior part of the nasopharynx of a marmot showing a bursa pharyngea homologous with that of man. It points backward and downward, however, rather than upward and backward, as in man (from Killian): *B.*, bursa pharyngea; *Lb.*, lip of the bursa; *E.*, epithelium; *Muc.*, mucous membrane; *Cst.*, constrictor pharyngeus muscle; *Sph.bas.*, basisphenoid; *S.sph.o.*, synchondrosis spheno-occipitalis; *Occ.bas.*, basioccipital bone; *A.*, arch of atlas, and *D.*, odontoid process of axis.

In 1895 Nebelthau [31] revived Froriep's view and pointed out that the contact of the pharyngeal mucous membrane with the notochord produces a fixation point at which the bursa pharyngea develops.

Five years later, Giuseppi Levi [32] reported that the notochord reaches the ventral aspect of the basal plate, courses for a certain distance in the

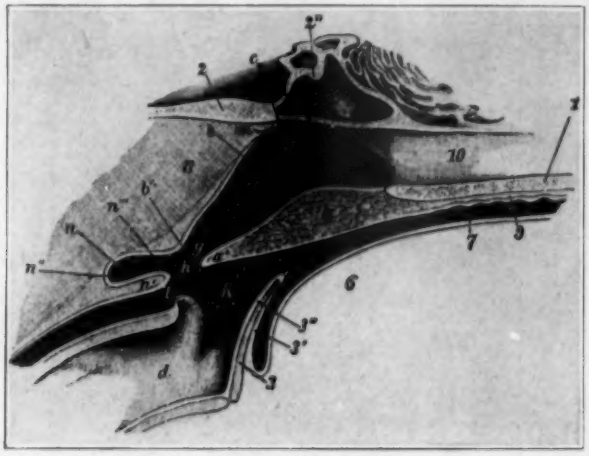

Fig. 34.—Median section of the pharynx of a pig (from Ellenberger and Baum: Handbuch der vergleichenden Anatomie der Haustiere, Berlin, A. Hirschwald, 1915, p. 397): *a*, velum; *d*, larynx; *g*, nasopharynx; *l*, esophagus; *n*, pharyngeal pouch; *n'*, ventral wall of pharyngeal pouch; *n''*, base of pharyngeal pouch, and *n'''*, opening of pharyngeal pouch.

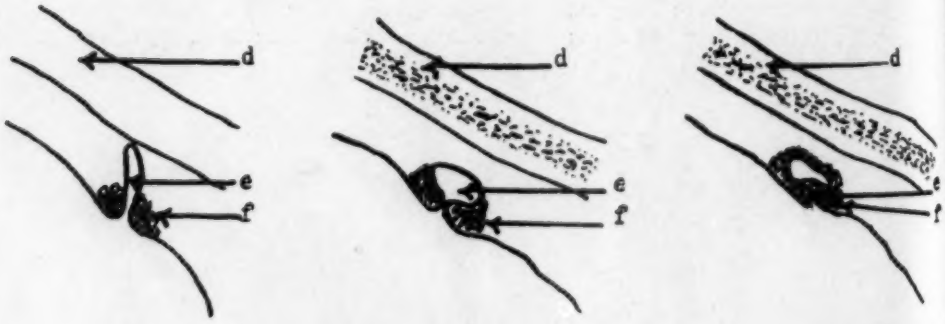

Fig. 35.—Diagrammatic presentation of Gradenigo's ideas as to the various forms of bursa pharyngea: *A.*, bursa pharyngea embryonalis or bursa pharyngea vera; *B.*, recessus pharyngeus medius; *C.*, pseudobursa or von Luschka's bursa; *d*, occipital bone or cartilage; *e*, bursa, and *f*, pharyngeal tonsillar tissue.

31. Nebelthau: Ueber die Gallertgeschwülste am Clivus Blumenbachii, Inaug. Diss., Marburg, 1895; quoted by Linck: Ztschr. f. Ohrenh. **62**:158, 1911.

32. Levi, G.: Beitrag zum Studium der Entwicklung des knorpeligen Primordialcraniums des Menschen, Arch. f. mikr. Anat. **55**:341, 1900.

perichondrium and then passes into the retropharyngeal connective tissue to lie in intimate contact with the epithelium of the bursa pharyngea.

In 1901, Gradenigo,[33] in a comprehensive work on hypertrophy of the pharyngeal tonsil, expressed his views on the pharyngeal bursa, of which the following summary is given:

Von Luschka described his bursa as it exists in adults, but also studied a similar structure in the fetus. Many of von Luschka's illustrations do not show in adults true bursae but pseudobursae, the result of pathologic changes in the pharyngeal tonsil; in fact, Gradenigo said that a true bursa exists only in the embryo. Gradenigo distinguished

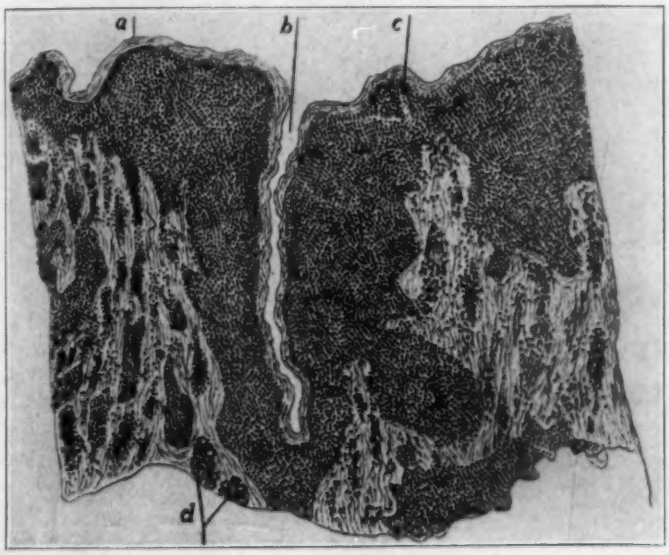

Fig. 36.—Contrast between the shape of the rounded bursa pharyngea and that of the normal median recess of the pharynx (from Disse, in Haymann: Handbuch der Laryngologie und Rhinologie, Vienna, Alfred Hölder, 1899, vol. 2, p. 24). The median recess in this picture is a mere slit. *a* represents the epithelium; *b*, the median recess of the pharynx; *c*, the tunica propria permeated with round cells, and *d*, glands.

the following kinds of bursae or depressions at or near the site of the bursa mentioned by von Luschka:

1. Bursa pharyngea embryonalis or bursa pharyngea vera, a small distinct cavity, which although not constant, appears in the embryo before the pharyngeal tonsil does. Its location is the posterior end of the primitive median raphe; it represents after the appearance of the pharyngeal tonsil the median furrow of

33. Gradenigo, G.: Die Hypertrophie der Rachentonsille: Eine Monographie, Klin. Vortr. d. Otol. u. Pharyngorhin. **4**:179, 1901.

this structure, and the essential character is that its blind end is directed backward against the basilar portion of the occipital bone and penetrates into the basilar fibrocartilage.

2. Recessus pharyngeus medius which results from the flattening of the primitive embryonal bursa. It lies, as does the primitive bursa, at the posterior end of the middle furrow of the tonsil; it is not made up of a true bursa or cavity, however, but of a deepening which does not reach the basilar fibrocartilage.

3. Pseudobursa or von Luschka's bursa, which bears a variable relationship to the midline. It is a cavity of variable size and position, which arises secondarily from the accumulation of secretion, during the process of involution by adhesions at more or less extensive areas at the edges of the split or at the middle furrow of the pharyngeal tonsil (fig. 35 *C*).

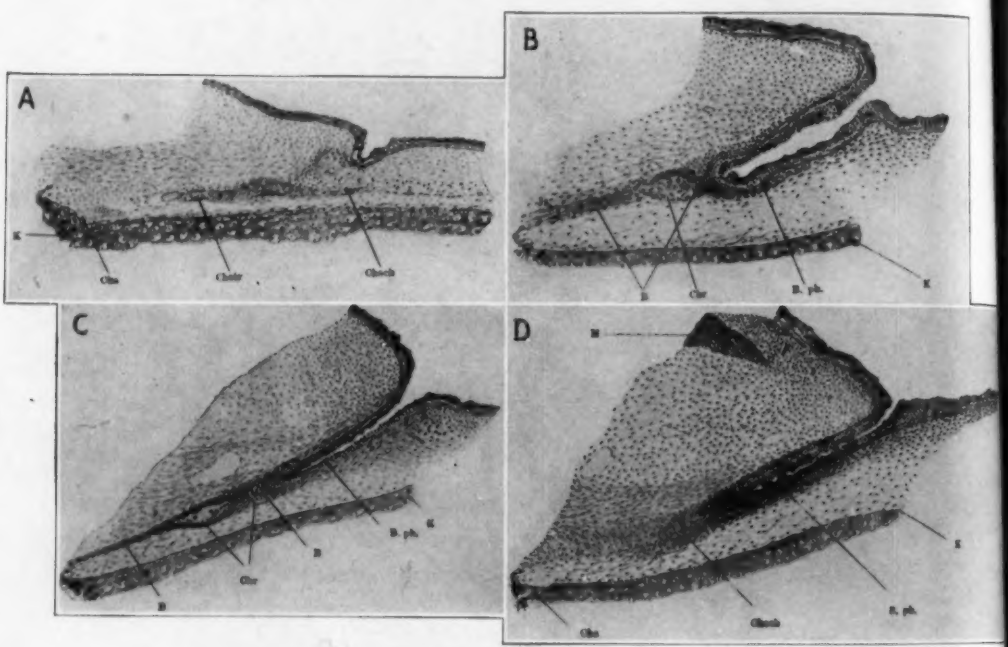

Fig. 37.—*A*, section showing flat groovelike appearance of bursa pharyngea embryonalis with a collection of cells of notochord (*Chsch*) at its apex; *B*, section showing a deeper and wider bursa pharyngea embryonalis the apex of which is connected with connective tissue strands (*B*) to exit of notochord; *C*, section showing a deep and narrow bursa pharyngea embryonalis the apex of which is connected with connective tissue strands (*B*) to the notochord (*Chr*). There is a long oval collection of vesicular cells attached to the bursa by means of fibrils. Small groups of notochord cells are seen between the fibrils; *D*, section to show a deep tubelike bursa pharyngea embryonalis with a homologous collection of notochord cells (*Chsch*) at the apex. These cells are connected with the apex of the bursa by bundles of fibrils (from Linck: Ztschr. f. Ohrenh. **62:**158, 1911). *K* indicates cartilage; *Cha*, site of exit of notochord; *Chstr*, cord of notochord; *Chsch*, notochord cells at apex of bursa; *B*, perichordal connective tissue strands; *Chr*, cells of notochord; *B.ph*, bursa pharyngea, and *M*, superior constrictor muscle of the pharynx.

Gradenigo's conception of the three kinds of bursae is shown in the accompanying sketches (fig. 35). As a matter of fact, the true median recess of the tonsil is often a mere slit, as is shown in figure 36.

These views of Gradenigo that there is a true bursa in embryonal life only and that von Luschka's bursa is a secondary product, the result of pathologic changes in the pharyngeal tonsil, are not held as correct at present, although it is probable that a true bursa, when it is present

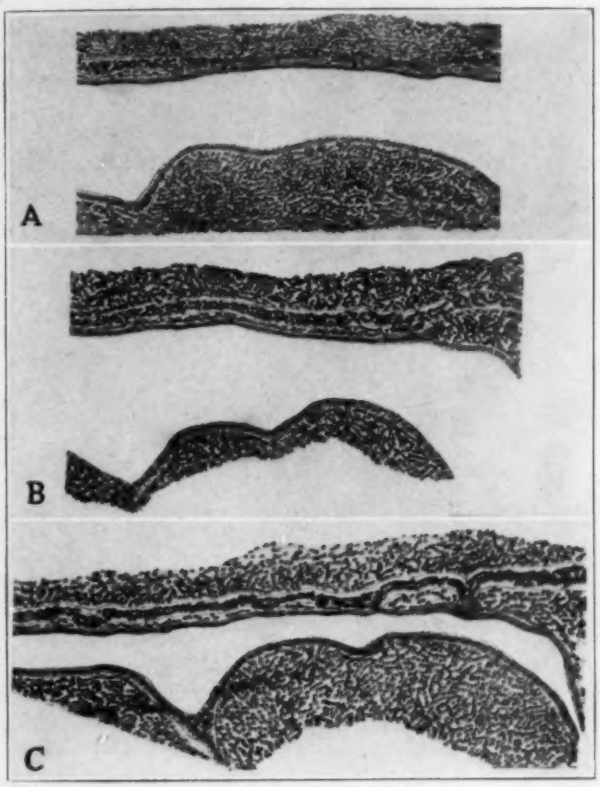

Fig. 38.—*A*, vault of pharynx and dorsum of tongue (6.6 mm.). Notochord in contact with pharyngeal epithelium near upper right portion of figure; × 100; *B*, vault of pharynx and dorsum of tongue (7.5 mm.); notochord is in distinct contact with pharyngeal epithelium, which is thickened in area of contact; × 100; *C*, vault of pharynx and dorsum of tongue (8 mm.); notochord makes distinct loop (upper right hand portion of figure) and comes in contact with the pharyngeal epithelium. Other less distinct points of contact of chorda and the pharyngeal epithelium shown; × 100 (these figures are taken from Huber: Anat. Rec. **6**:373, 1912).

in postembryonal life, may undergo a certain amount of flattening. It is not certain that von Luschka really studied the bursa in the embryo, at least this is not shown by the data immediately at my disposal. His

reference to his treatise on the pharynx is merely to an embryo in which he was studying the invagination of the pharynx (Rathke's pocket) that went to form the anterior lobe of the hypophysis.

Further studies on the development of the bursa pharyngea were carried on by Gage [34] (1906), who found that in human embryos the notochord lies directly in contact with the roof of the primitive pharynx. Her studies included several embryos of different species of animals of

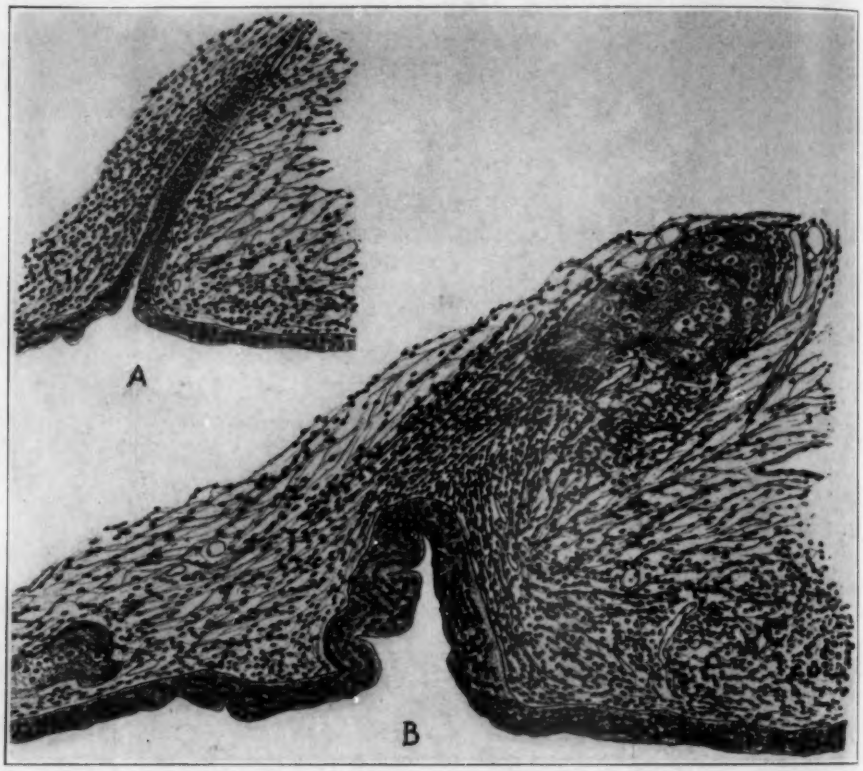

Fig. 39.—*A*, the deep pharyngeal bursa and surrounding retropharyngeal mucosa (32 mm.); upper portion of figure shows a thickened chordal sheath accompanied by parallel strands of fibrous tissue, reaching the blind end of the bursa; × 100; *B*, vault of pharynx in region of the pharyngeal bursa (60 mm.). Large mass of notochordal tissue dorsal to bursa and united to bursa by strands of fibrous tissue having a parallel course. Smaller mass of chordal tissue, lying near pharyngeal epithelium, to the left in figure; × 100. (These figures are taken from Huber.)

which the only animal embryo showing relations comparable with those of man was that of the pig.

34. Gage, S. P.: The Notochord of the Head in Human Embryos of the Third to the Twelfth Week and Comparisons with the Other Vertebrates, Science **24**: 295, 1906.

In 1910, Mayer [35] identified the bursa with Seessel's pocket and denied that it was a passive invagination as it had been regarded previously by Killian.

Linck,[36] in 1911, concluded from his studies of human embryos that the bursa was the result of traction exerted by the notochord on the primitive pharyngeal mucosa, the former growing more rapidly than the latter, with which it comes in contact (fig. 37 *A*, *B*, *C* and *D*). He

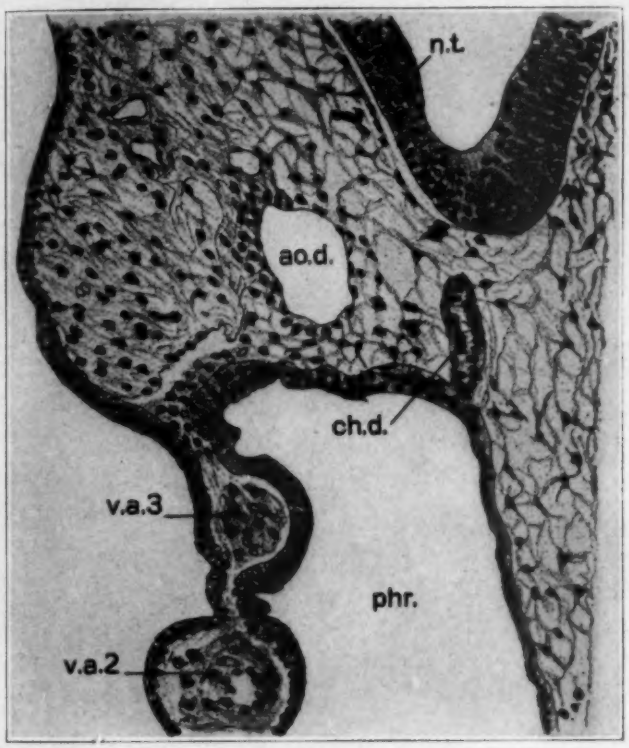

Fig. 40.—Section from human embryo of 24 somites, showing fusion of the chorda dorsalis to the entoderm of the digestive tract. In this specimen the fusion is the more anterior of two fissures and hence does not represent the site of the bursa pharyngea (from Johnson: Contributions to Embryology 19, publication 226, p. 125): *n.t.*, medullary tube; *ao.d.*, aorta dorsalis; *ch.d.*, notochord; *v.a.2*, second visceral arch; *v.a.3*, third visceral arch, and *phr.*, pharynx.

35. Mayer, R.: Ueber die Bildung des Bursa pharyngea medius s. Bursa pharyngea in Zusammenhang mit der Chorda bei menschlichen Embryonen, Anat. Anz. **37**:449, 1910.

36. Linck, A.: Ueber die Genese der Bursa pharyngea embryonalis, Ztschr. f. Ohrenh. **62**:158, 1911; Beitrag zur Kenntnis der menschliche Chorda dorsalis im Hals und Kopfskelet, etc., Anat. Hefte **42**:605, 1911.

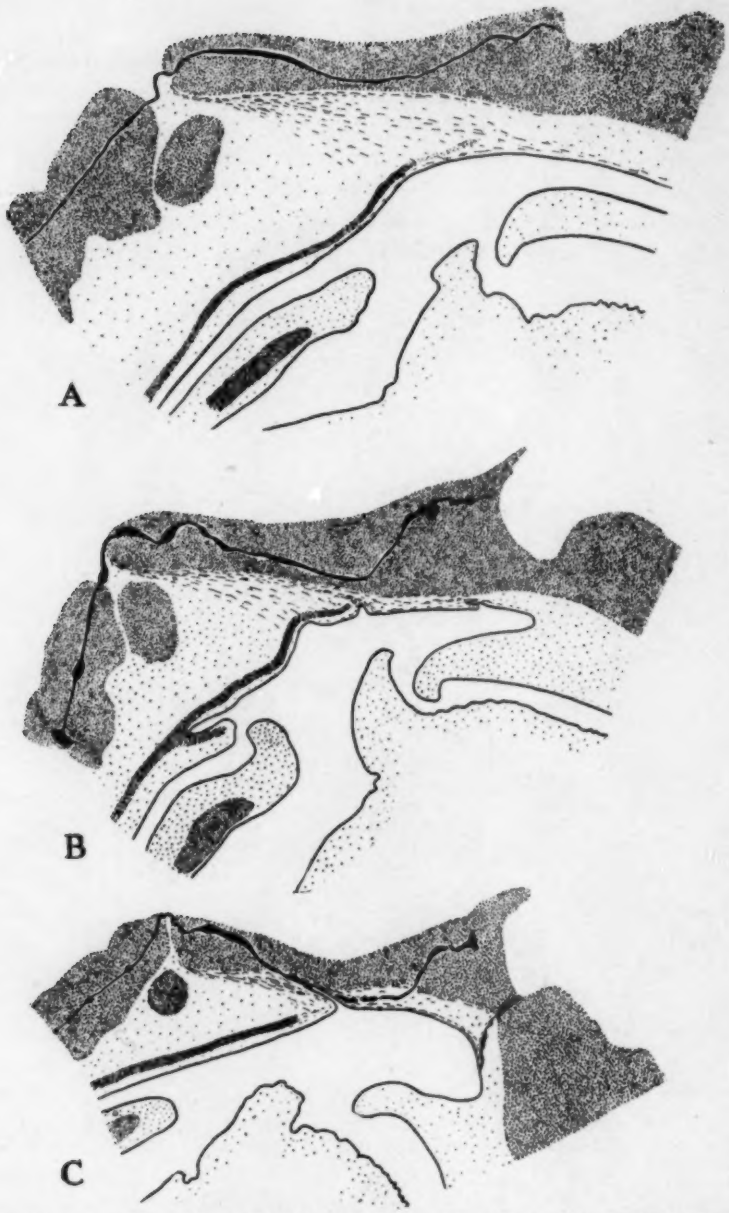

Fig. 41.—*A*, calf embryo, 62 mm. Reconstruction of median sagittal plane of pharyngeal region; × 10; *B*, pig embryo of 50 mm. total length. Reconstruction of median sagittal plane of pharyngeal region, showing two small outpocketings of pharyngeal epithelium related to strands of the fascia pharyngobasilaris; × 10. Note that in the pig the notochord does not leave the basilar plate; *C*, human embryo 46 mm. in crown-rump length. Reconstruction of median sagittal plane of pharyngeal region, showing a large bursa, the notochord and persistent stalk of the hypophysis. The fascia pharyngobasilaris is also indicated; × 10. (All three sketches are from Atterbury: Anat. Rec. **13**:467, 1917; **16**:251, 1919).

also mentioned two factors which produced this bursa: (1) traction exerted by connective tissue bands which replace the notorchord, and (2) active ingrowth of the pharyngeal epithelium which becomes funnel-shaped.

Huber [37] made extensive studies of human embryos and in 1912 reported that he found a distinct relation between the notochord and the bursa pharyngea. In embryos with a crown-rump length of from 5 to 8 cm. there is an area in contact with the pharyngeal entoderm at the termination of the ventral flexure of the notochord. This endoterm always shows an increase in the production of its cells (figs. 38 *A*, *B* and *C* and 39 *A* and *B*). In fact, the contact here may occur at several points. However, I am especially concerned with the most caudad of these points, a point that has a definite position for each of the embry-

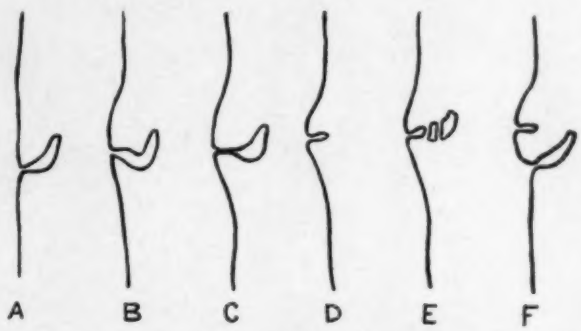

Fig. 42.—The relationships of the bursa pharyngea to the pharyngeal tonsil: *A*, bursa pharyngea, no tonsillar tissue; *B*, median recess of the tonsil communicating with the bursa pharyngea; *C*, same as *B*, except that the median recess in consequence of a pathologic process or otherwise, is a fine tube; *D*, median recess of the tonsil, with no bursa pharyngea; *E*, pathologic process in the tonsil has caused the formation of two membranes which separate the bursa from the median recess; *F*, the median recess and the bursa are distinct structures.

onal stages of development. This point, which is not seen in all human embryos, represents the site of the bursa pharyngea. Seessel's pocket plays no part in the production of the bursa. Huber also doubted the influence of the fascia pharyngobasilaris as a factor in the production of the bursa.

Watts [38] confirmed the view of Huber. He noted in very young twin embroys the lateness of the development of the notorchord at the

37. Huber, G. C.: On the Relation of the Chorda Dorsalis to the Anlage of the Pharyngeal Bursa or Median Pharyngeal Recess, Anat. Rec. **6**:373, 1912.

38. Watts, J. C.: Description of Two Young Twin Human Embryos with 17-18 Paired Somites, Contrib. Embryol., no. 2, Carnegie Institution of Washington, 1915, publication 222.

site of the future bursa pharyngea. Cordal contacts with the pharyngeal entoderm were noted by Johnson,[39] from whose work figure 40 is taken.

Atterbury [40] found that in the pig embryo there is a series of pockets that correspond with the bursa pharyngea in man. She also pointed out that these arise independent of contacts of the notochord with the pharyngeal entoderm, and that the growth of the fascia pharyngobasilaris acts as the essential causative factor. Later, she demonstrated the relation of this fascia to certain outpocketings of the pharyngeal entoderm in the calf embryo and showed that in the human embryo the traction exerted by the connective tissue sheath of the notochord acts as one of the factors that produce the bursa pharyngea. Some of her illustrations are shown in figure 41. It will be noted that the human embryo is the only embryo in which the notochord leaves the basilar cartilage.

SUMMARY

This survey covers the most important work done to explain the development of the bursa pharyngea. It is evident from what has been said that this bursa is not the same structure as that called Seessel's pocket or as that called Rathke's pouch. Huber has definitely established this fact, although Radford,[41] basing his investigation on the ferret embryo, still doubted this evidence and identified the bursa with Seessel's pocket. Clinically, when the bursa occurs, its position is characteristic; it occupies a position in the midline of the pharynx just anterior to the upper border of the superior constrictor muscle. In cases in which the pharyngeal tonsil is present, the bursa bears an intimate relationship to this structure, a relationship that is represented graphically in figure 42.

Cysts and neoplasms of the nasopharynx demand careful consideration to determine their respective origin. Cysts of the bursa pharyngea were discussed by von Luschka in his classic work on the pharynx. He referred to von Troeltsch [42] and Czermak,[43] who reported cysts that

39. Johnson, F. P.: A Human Embryo of Twenty-Four Pairs of Somites. Contrib. Embryol. no. 19, Carnegie Institution of Washington, 1917, publication 226, p. 125.

40. Atterbury, R. R.: Bursa and Tonsilla Pharyngea: A Note on the Relations in the Embryo Calf, Anat. Rec. **16**:251, 1919; On the Relation of the Head Chorda to the Pharyngeal Epithelium in the Pig Embryo: A Contribution to the Development of the Bursa Pharyngea and the Tonsilla Pharyngea, Anat. Rec. **13**:467, 1917.

41. Radford, M.: A Note on the Development of Pharyngeal Bursa in the Ferret Embryo, Anat. Anz. **44**:371, 1913.

42. von Troeltsch: Anatomische Beiträge zur Ohrenheilkunde, Virchows Arch. f. path. Anat. **17**:1, 1859.

43. Czermak, quoted by Semeleder, F.: Die Rhinoskopie und ihr Werth für die aerztliche Praxis, 1862, p. 54.

were undoubtedly cysts of the bursa. Several cases of cysts of the bursa (fig. 23) have been reported in the literature. Thus Loewenberg [44] gave a graphic description of one in 1881. Cysts of the nasopharynx may arise from other structures than the bursa.

Clinically, the bursa pharyngea is of special interest. In the eighties and nineties its diseases were the subject of heated discussions. In 1885, Tornwaldt [26] of Dantzig reported cases of inflammatory changes of this bursa and others in which there was cyst formation. He also mentioned cases in which he observed discharges from what he assumed to be the bursa pharyngea. Most of the cases with a diseased bursa reported by Tornwaldt were associated with other systemic disturbances which he thought were caused by the diseased condition of the bursa pharyngea. When his opinion is considered in the light of modern medicine, one finds that in spite of errors due to overenthusiasm he was anticipating the era of focal infection. He undoubtedly awakened interest in a neglected field of human anatomy and brought to the attention of physicians a careful study of diseases of the pharynx.

Cysts of the nasopharynx should be carefully examined microscopically. It is probable that most of the larger cysts of this region the linings of which contain ciliated epithelium have been cysts of the bursa pharyngea. It is true that some of these cysts may be of the dermoid type. The histopathologist examining these lesions, however, should consider in his investigation the embryology of the pharynx.

CONCLUSIONS

In conclusion the following statements are submitted:

1. A true bursa pharyngea in adults is an independent structure and is not to be regarded simply as the median recess of the pharyngeal tonsil.

2. It does not develop from Seessel's pocket.

3. It does not develop from Rathke's pouch.

4. A true bursa pharyngea occurs somewhat frequently in man during embryonal life; it takes its origin from adhesions of the notochord to the pharyngeal entoderm, and its production may be aided in the general process of growth by the traction of fibrous bands, of the fascia pharyngobasilaris or of both, as well as by active growth of the entoderm.

5. The bursa has a definite location for each age of the embryo.

6. The true bursa has a definite location in adults: above the upper border of the superior constrictor muscle of the pharynx.

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44. Loewenberg: Les végétations adénoides dans la voûte du pharynx, Tr. Internat. Med. Cong., London, 1881, p. 281.

7. The true bursa in adults does not in all cases present merely a "flattening" or shortening of the bursa in the embryo. In certain cases it may extend to the occipital bone and indent that structure.

8. It is probable that the bursa in adults represents the persistence of the embryonal bursa.

9. The bursa in adults may be the seat of inflammation and cyst formation.

10. The bursa is an independent structure which does not bear any relation to cleft palate.

The study of the bursa pharyngea, of which a summary has been presented in this paper, has increased the realization of the complexity of the structures that go to form the nasopharynx.

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HISTOPATHOLOGY AND BACTERIOLOGY OF SINUSITIS

WITH COMMENTS ON POSTOPERATIVE REPAIR *

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Owing to the increasing recognition of the importance of sinus infection in relation to respiratory and systemic diseases, during the last few years there has been a renewed interest in the pathology and bacteriology of sinus infections and also in the end-results of the various methods of treatment. These are the fundamentals on which any therapy must be based.

Among many others, my co-workers and I have been carrying on investigations along these lines during the past four years at the University of Oregon Medical School. Whether this research brings out anything new, or simply serves to refresh our memory and clarify our ideas of what is already known, we think it may be useful. Three previous reports have been presented, and what we now offer is the result of further research into some of the problems considered before.

POSTOPERATIVE REPAIR IN THE SINUSES

During the last two or three years there have appeared several reports of investigations of healing or repair after operation on the sinuses. Some of these present the observations on animals after operation, some on biopsies from patients and some on postmortem material.

Our observations are based on tissues secured from fifteen persons; seven specimens from patients on whom a complete operation was performed after a previous Caldwell-Luc operation, six biopsies obtained from antrums through the counteropening, one specimen from an antrum through a large permanent opening in the canine fossa and one from a healed ethmoidal sinus. In general, we have found that a biopsy specimen gives meager information. Conclusive evidence was obtained from the cases in which a second operation was performed and in which the whole lining was removed and a complete series of sections made.

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* From the Department of Otolaryngology and the Department of Pathology of the University of Oregon Medical School.

In a previous report [1] we showed that the new formed submucosa is typical scar tissue and is histologically different from the original sinus mucosa (fig. 1). On casual inspection the two seem similar, but microscopic analysis with Weigert's, van Gieson's and Mallory's stains brings out the shades of difference.

We believe that the nasal mucous membrane plays an important part in determining the character of the tissue that will be found in a healed sinus after operation. It is of particular significance as a source of the new epithelium that will cover the scar tissue (fig. 2). When the source of the new epithelium is in the nose, it will retain to some extent the character of the nasal epithelium, but when there is a possibility of

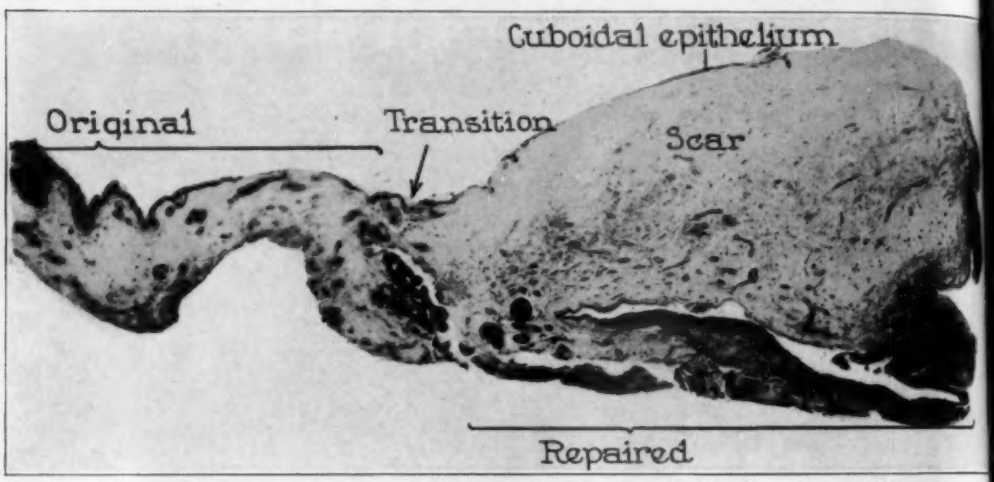

Fig. 1.—Repair in the mucous membrane of the antrum. At a previous Caldwell-Luc operation only the anterior third of the lining was removed. Two years later another operation was performed and the whole lining removed. A portion of the original mucosa with its glands, blood vessels and ciliated columnar epithelium appears on the left; on the right is pure scar tissue. Note the absence of glands and the character of the simple blood channels characteristic of scar tissue. The transition from sinus mucosa to scar tissue is definite; Zenker's fixation, hematoxylin-eosin stain; reduced from × 8.

its extending in from some other source, such as the buccal cavity, it has the characteristics of buccal epithelium. The spread of nasal epithelium is not reliable and is inconstant, the character changing from stratified ciliated columnar cells to a single layer of flat cuboidal cells within the same cavity as late as two years after operation.

1. Semenov, H., and Kistner, F. B.: Repair in the Paranasal Sinuses of Man Following Removal of the Mucous Membrane Lining, Proc. Soc. Exper. Biol. & Med. **27**:332, 1930.

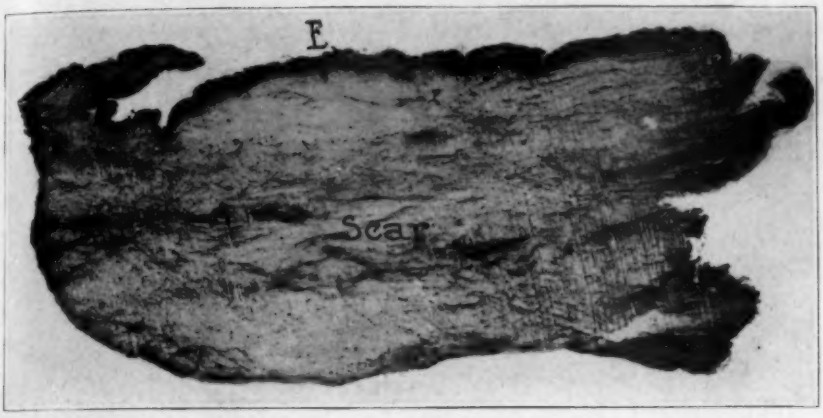

Fig. 2.—Repair in the anterior wall of the antrum (from the region of the canine fossa). A thick deposit of organized scar tissue covered by ciliated columnar stratified epithelium is seen. The epithelium (*E*) has spread in from the nasal mucosa. No glands, arteries or other typical structures of sinus mucous membrane have appeared. Radical exenteration was performed, followed by a second operation twenty months later. Zenker's fixation, hematoxylin-eosin stain; × 8.

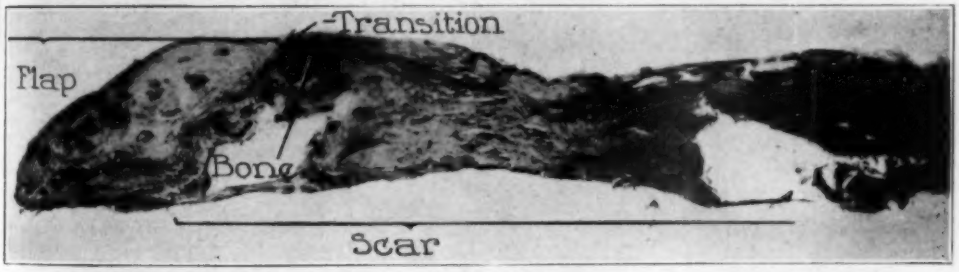

Fig. 3.—Photomicrograph of a sinus on which a second operation was performed, showing the medial half of the new lining as it appears twenty months after a previous radical exenteration had been done. This section reveals a transition from nasal flap to scar tissue: on left, flap, then white fibrous connective tissue, and on right, bone. The submucosa of the flap contains the usual nasal glands and the connective tissue stains relatively pale. The new lining is solid scar tissue and contains no glands or blood vessels. Numerous irregular cavities on the lower surface are due to the new formation of bone; a few spicules lie embedded in the scar. The transition between flap and scar is abrupt. Zenker's fixation, van Gieson's connective tissue stain; × 8. (Subsequent illustrations show characteristic structures under higher magnification.)

That the histologic observations in healed sinuses will vary according to the type of operation or the thoroughness of exenteration is manifested by an analysis and comparison of the tissues that we removed. For instance, in cases in which a second operation was performed, as we had good reason to believe that the first operation was not thorough, we found fragments of the original mucous membrane still attached to

Fig. 4.—The transition between nasal flap and scar tissue lining the antrum. Note that the nasal epithelium ends abruptly by curling downward. The scar is totally devoid of epithelium beyond this point. The line of cleavage between scar tissue and nasal transplant is indicated and the stratiform character of the white fibrous scar tissue can be discerned. The dark mass to the right is a spicule of new-formed bone; part of the scar. Zenker's fixation, hematoxylin-eosin stain.

the bone or merged with the surrounding scar tissue. Apparently these areas were not disturbed at the time of the first operation. Tonndorf [2] made the same observation in a case that came to autopsy four weeks

2. Tonndorf: Beiträge zur Ausheilung der nach Luc-Caldwell operierten Kieferhöhle, Ztschr. f. Hals-, Nasen- u. Ohrenh. **22**:54, 1928.

after a Caldwell-Luc operation, and stressed the improbability of removing all of the mucous membrane by curettage. Subperiosteal clusters of glands may cling to the bone and appear in the scar after operation. Examination of the tissues filling the opening in the canine fossa in cases in which a second operation has been performed, when one is sure that the entire thickness of the original mucous membrane

Fig. 5.—Photomicrograph with a higher magnification showing the character of the tissue drawn into the antrum, the nasal flap shown in figure 3. Here there are nasal epithelium, basal membrane, gland ducts, glands, erectile tissue, true vascular channels and underneath it all is scar tissue, indicating where the transplant has been grafted and fused with the new lining. Weigert's elastic tissue stain shows the characteristic fibrils as they appear in nasal mucous membrane and thus further substantiates the evidence that this highly organized structure did not regenerate but was transplanted into the sinus fully developed. Zenker's fixation, hematoxylin-eosin stain.

has been destroyed, reveals a simple scar tissue covered with stratified ciliated columnar epithelium and shows a complete absence of glands (fig. 2).

In sections made from tissues near the opening into the nose we often find the characteristic structure of nasal mucous membrane. We believe that this is nasal mucous membrane transplanted into the antrum either intentionally or accidentally; for its characteristic appearance disappears abruptly when one reaches the new-formed tissues lining the rest of the cavity (fig. 3). Such fragments of nasal mucous membrane retain all of the characteristic structures of nasal mucosa (fig. 5). The histologic analysis of such material reveals erectile tissue, glands, large arteries with smooth muscle walls and the elastic tissue fibrils of nasal mucosa. An abrupt dividing line between the transplant of nasal mucosa and the remainder of the new-formed lining can be demonstrated

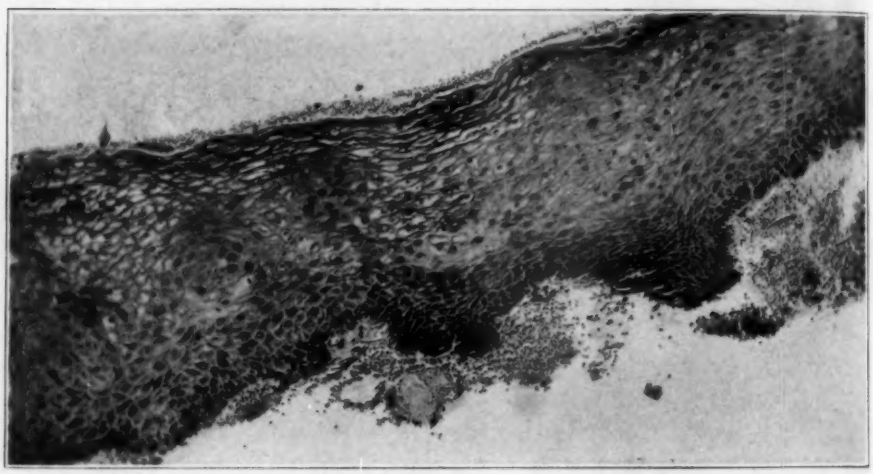

Fig. 6.—Photomicrograph showing stratified squamous epithelium removed from the posterior superior wall of an antrum one year after exenteration of the original lining. This is oral epithelium and demonstrates that the new lining does not regenerate but spreads into the operated cavity from the adjacent mucous membrane. Formaldehyde, hematoxylin-eosin stain.

(figs. 3 and 4). The latter is white fibrous scar tissue and instead of arteries has numerous irregular capillary channels.

The foregoing observations concern the types of repair in which epithelization takes place from the epithelium of the mucous membrane remaining in the sinus or from that of the nasal cavity. We now consider a third type found in a patient who presented himself for operation with a large permanent opening in the canine fossa which exposed the antrum. One year after exenteration, the opening into the buccal cavity having remained patent, we were able to remove a large biopsy specimen from the upper posterior portion of the cavity. The new lining was found to be covered with a thick layer of stratified squamous

epithelium (fig. 6). It undoubtedly extended into the antrum from the buccal cavity by way of the permanent opening. This was the only instance in specimens from fifteen persons in which we found stratified squamous epithelium.

HYPERPLASTIC SINUSITIS

I have expressed the belief [3] that hyperplastic sinusitis is the latent stage of sinus infection, that in its early stages it is usually purulent, but that it may be the result of an attenuated or low grade infection on an allergic basis without ever showing evidence of purulency. The history, the surgical specimens and the histopathologic reconstruction of an ethmoidal cell from a clinical case carefully observed for a period of nine months to some extent confirm this belief.

REPORT OF A CASE

Mr. L. T., aged 28, had had frequent colds for five years, and when he presented himself had fever, pain and swelling over the left frontal sinus. Clinical,

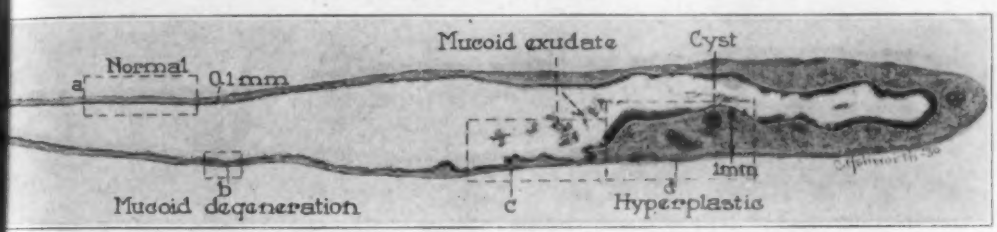

Fig. 7.—Hyperplastic ethmoidal sinus showing a cross-section of the entire cell as it appears drawn to scale (circumference 40 mm.). The histopathologic observations are indicated and the micrometer measurements noted. The following photomicrographs reveal the salient features of this cell. Formaldehyde, hematoxylin-eosin material; reduced from × 10.

laboratory and roentgen evidence pointed to a purulent left pansinusitis. Under conservative treatment the condition subsided, but did not disappear. Headache, vertigo, tenderness in the left frontal region and a moderate purulent discharge continued for three months, so it was decided to perform an intranasal frontal drainage. The patient was kept under continual observation for the next six months the distress and discharge continued in spite of the previous treatment. The patient was advised to have an external frontal ethmoidectomy. At operation, 260 days after the purulent sinusitis appeared, a large frontal sinus filled with mucopus and lined by a thick, soft, rough and dark red membrane was found. The immediate ethmoidal cells were opened and found to be definitely hyperplastic but devoid of secretion.

Here two types of pathology were present: a purulent frontal, and a hyperplastic ethmoidal, sinusitis in contiguous sinuses, on the same side.

3. Kistner, F. B.: Chronic Nonpurulent Sinusitis and Its Clinical Significance, Ann. Otol. Rhin. & Laryng. **38**:795, 1929.

One can reasonably assume from the appearance of the nose and the history that the ethmoidal cells were at one time purulent. It is interesting to note that only a part of the ethmoidal mucous membrane was hyperplasic. From the appearance of serial sections of this sinus, as shown in figure 7 which is a cross-section of the whole cell, it was evident that part of the lining had undergone successful resolution while the glandular portion had become cystic and undergone a hyperplastic degeneration. Measurement with a micrometer showed the former to be

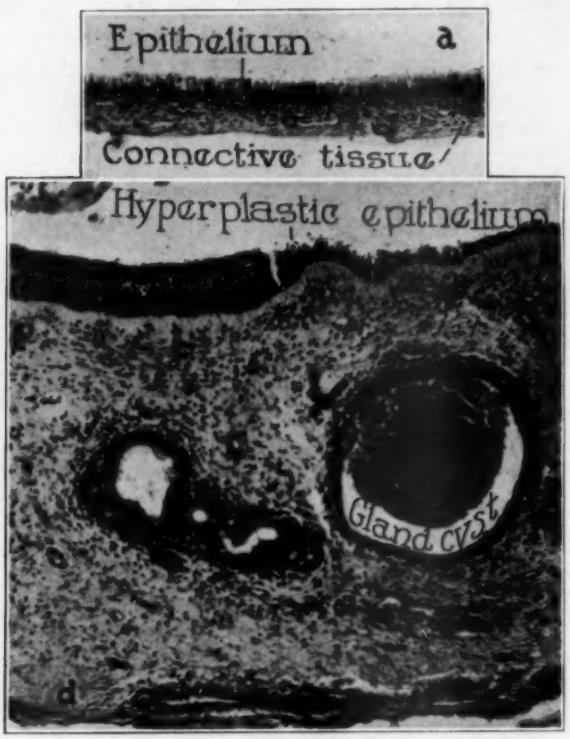

Fig. 8.—Photomicrographs in juxtaposition taken from areas *a* and *d* of figure 7, showing the normal and hyperplastic. Note the thickness of the epithelium and submucosa. Observe the cystic degeneration of the glands. Formaldehyde, hematoxylin-eosin stain; × 150.

0.1 mm. in thickness, or practically normal, while the latter was 1 mm. thick.

Microscopic study of the normal-appearing area at point *a* of figure 7 revealed a single layer of ciliated columnar epithelium with a hyalinized basement membrane and a thin tunica propria. The hyalinization of the basal membrane is evidence of a previous inflammatory process. As we circled around the lining we found an irregular folding and

formation of goblet cells at point *b;* at *c* mucoid degeneration of the epithelium, edema and leukocytic infiltration of the submucosa became marked. A chain of streptococci was also observed at this point. At *d* the pathologic picture was in full bloom; the epithelium showed at least eight layers of cuboidal cells piled on top of one another covered by columnar cells with very short cilia. Figure 8 is a photomicrograph

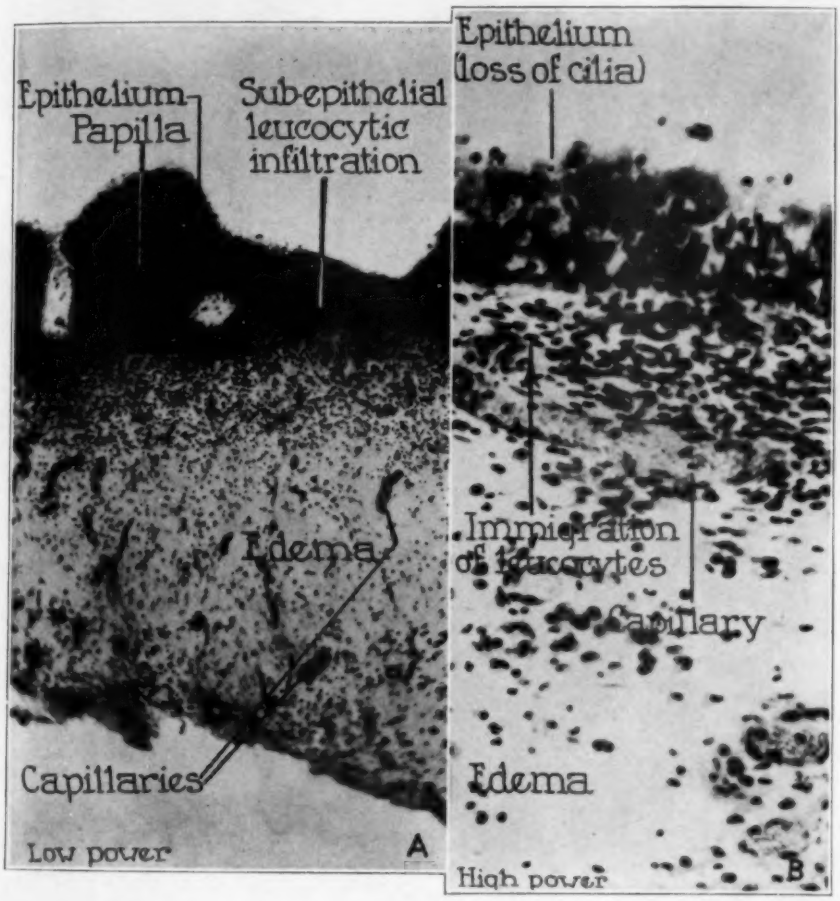

Fig. 9.—*A,* purulent frontal sinus from the same case. Note the dense subepithelial accumulation of leukocytes and the papillary folding of the surface. There is much edema, capillary proliferation and dilatation. Formaldehyde, hematoxylin-eosin stain; × 150. *B,* epithelium from same purulent frontal sinus. Note the capillary dilatation, edema, plasma cells, and diapedesis of leukocytes toward the epithelium. The cilia of the epithelium are degenerated, and there is an early hyperplasia of the cells. Formaldehyde, hematoxylin-eosin stain; × 500.

made to scale, showing the normal area *a* in direct juxtaposition to the hyperplastic area, so that the shades of difference between them are

obvious. One may also note the hyaline hypertrophy of the basement membrane *d* and the dilated degenerating glands. A further noteworthy feature of chronic hyperplastic sinusitis is the leukocytic infiltration which in this case consisted almost entirely of eosinophils and plasma cells. It is interesting to observe that the hyperplastic epithelium of itself is as thick as the entire layer of normal epithelium and submucosa combined (fig. 8).

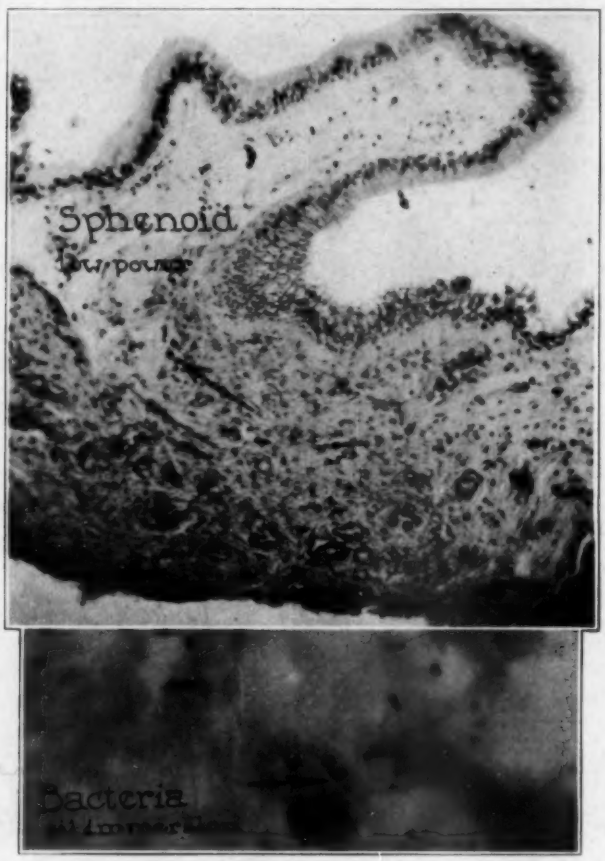

Fig. 10.—Photomicrographs showing a hyperplastic sphenoid under low power magnification and an oil immersion of the bacteria found at the point indicated by the arrow. From a case of retrobulbar neuritis.

The histopathologic process in the frontal sinus was of the usual chronic purulent type with marked edema and polymorphonuclear and plasma cell infiltration under the epithelium. In figure 9 is seen a photomicrograph taken with the same magnification as was used for figure 8; thus the degree of thickening is apparent when one contrasts

it with the normal portion (*A*). Although the subepithelial tissues are densely infiltrated by leukocytes and the greatest reaction is in and under the epithelium, nevertheless the infection is deep-seated. As I previously showed [4] and as this case further demonstrates, drainage is adequate in some cases.

BACTERIOLOGY

Although hyperplastic sinuses are not suppurating they are usually infected with pathogenic organisms, the streptococcus predominating.

Fig. 11.—*A*, photomicrographs (taken by Dr. E. C. Rosenow) of hyperplastic antrum in a case of arthritis, showing the low power field and diplostreptococci found at the points indicated by the arrow and crosses. The bacteria are most abundant in the depths of the mucous membrane. *B*, bacteria in depth of tunica propria. *C*, bacteria near periosteal layer.

The tissues removed from patients operated on were washed, ground in sterile sand and cultures or inoculations into rabbits were made. Cul-

4. Kistner, F. B.: Pathology of Sinusitis, Ann. Otol. Rhin. & Laryng. **38**: 977, 1929; Tr. Pacific Coast Oto-Ophth. Soc., 1929.

tures were made from more than 400 sinuses, and a mixture of bacteria was usually found in each case. A pure culture of one organism was seldom found in cases of chronic sinusitis. In these tissues the streptococci were the most constant, being present in 94.5 per cent of the sinuses; 35 per cent of these were hemolytic B, 33 per cent hemolytic green-producing or A, 14 per cent *Streptococcus viridans* and 18 per cent nonhemolytic streptococci of the gamma type. In addition to, and in combination with, the streptococci we found in the order of their frequency: staphylococci, *Micrococcus catarrhalis,* pneumococci, Friedländer's bacilli, influenza bacilli, colon bacilli, diphtheroids and *Streptothrix.*

We have been able to stain the organisms in the tissues in the same relative numbers as we recovered them in the cultures. Tunis [5] stained streptococci in the wall of a cyst from a patient with empyema of the antrum. His tissues were taken post mortem. Watson-Williams [6] also stained bacteria in the tissues from a chronic purulent antrum and remarked on the tendency to morphologic degeneracy of the organisms, as a result of diminished virulency and subjection to the tissue defenses. As stated, both of these men were working with membranes from purulent sinuses. Our investigation has been confined to chronic latent or nonpurulent sinusitis. Tissues removed from the antrums and sphenoids yielded the same types of bacteria. In figure 9*B* is seen a hyperplastic sphenoid in which diplostreptococci were stained in the depths of the tunica propria.

At his request, we sent to Dr. Rosenow, of the Mayo Foundation, some of our tissues including a number of specimens of hyperplastic or nonpurulent sinusitis. The following is a quotation from his letter reporting the results of his examination:

> I am sure you will be interested in the fact that I have found unmistakable organisms in all of the sections of the different cases which you sent me. The bacteria were found to be by far the most numerous in the deeper layers, especially next to the periosteum, less numerous in particularly edematous areas with relatively slight cellular infiltration peripheral to areas of round cell infiltration surrounding the deeper vessels and mucous glands, still less numerous immediately beneath the epithelial layer in the submucosa, within the epithelial layer itself and in the exudate in the folds of the mucous membrane; and they were found least often on the surface of the epithelial lining.
>
> In all sections gram-positive diplococci, certainly not staphylococci, were found. In most of them only gram-positive diplococci were found, but in a few scattered bacilli, resembling *Bacillus fusiformis* and one yielded staphylococci; according to the information sent me, staphylococci occurred in considerable numbers in the cultures.

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5. Tunis, J. P.: Inflammation of the Sinus Maxillaris with Special Reference to Empyema, Laryngoscope **20**:931, 1910.

6. Watson-Williams, P.: Remarks on Tissue Invasion of Low Grade Pyogenic Organisms, J. Laryng. & Otol. **144**:598, 1929.

CONCLUSIONS

1. The typical features of a hyperplastic ethmoid cell are presented. Hyperplasia in a sinus mucosa is one of the usual structural changes found in chronic latent or nonpurulent sinusitis. Whether it is of bacterial origin or due to an underlying allergy or vitamin deficiency, it exhibits epithelial or connective tissue proliferation. In patients in whom this type of sinusitis was associated with, or presumed to be a contributory factor in, a systemic disease, we found these tissues to contain pathogenic organisms.

2. The mucous membrane of the paranasal sinuses has a definite characteristic architecture. Even when modified by pathologic changes, it retains all of its structural elements. It should not be confused with nasal mucosa. The sinus and nasal mucous membranes differ greatly in histologic structure, organization and appearance. After complete destruction, the sinus mucous membrane does not regenerate its structures; instead there is a process of repair and bone proliferation. The walls of the cavity are lined by a layer of granulation tissue, variable in thickness, which eventually organizes and forms typical scar tissue. This becomes covered by an epithelium of variable character, determined by its place of origin and influence by its distance from the ancestral epithelium.

IMMATURE WHITE BLOOD CELLS IN OTOLOGIC INFECTIONS *

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The most common laboratory procedure for patients suffering from an otologic infection that is suspected of having passed beyond the confines of otitis media is the total leukocyte count, supplemented frequently by a differential estimation. The information expected is an indication of the severity of the infection and some knowledge of that vague and complex factor in illness which is termed "resistance." This is a procedure with years of priority. In general, a high count has been thought to mean a severe infection with a good resistance; a low count in the presence of a substantial infection, a poor resistance in the majority of diseases in which fever is a prominent symptom. These interpretations are not without error, however, for the white cell count may be found within normal limits and often only moderately elevated in patients who give clinical evidence of a severe toxemia but who manifest good resistance; the differential count may likewise be misleading.

These limitations of the white blood cell picture have long been a matter of common experience. Twenty-five years ago, Arneth, a hematologist, offered an analysis of the differential leukocyte picture interpreted to give more accurate data on the presence of infectious processes, their severity and prognosis. Based on the assumption that the toxic stimulus of an infection of the bone-marrow brought immature neutrophils into the blood stream, he classified the neutrophils in five groups, each group representing a different degree of maturity estimated from the extent of segmentation of the nucleus. The presence and number of immature neutrophils above the normal were considered indicative of the intensity of an infection. The complexity of this classification made it an impracticable procedure even for a hematologist. In 1911 Schilling offered a modification of Arneth's idea, which in subsequent studies has made the Schilling index popular.

Schilling modified the interpretation of the ordinary differential method by considering three classes of immature neutrophils. The most immature form is represented by the true myelocyte. This is not

* Submitted for publication, Aug. 14, 1930.

* From the Oto-Laryngology Service of the Massachusetts Eye and Ear Infirmary.

found in the normal blood. A little "older" neutrophil is the young metamyelocyte, in which the nucleus is beginning to be indented or is elongated; this form is also absent from the normal blood. A neutrophil with the nucleus in a bandlike or rodlike arrangement, which often assumes a horseshoe form, represents a stage of maturity preceding the mature nucleus, which is segmented and connected with each lobe by a slender thread of chromatin. Figure 1 is a schematic representation of these nuclear forms. If there is a definite band of chromatin connecting the different parts of the nucleus, the neutrophil cannot be said to have reached full maturity. In the accompanying photomicrograph

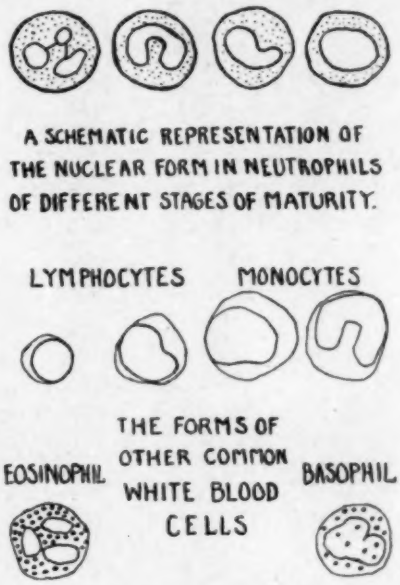

Fig. 1.—Nuclear forms seen in neutrophils.

(fig. 2) are illustrations of the most common forms of immature neutrophils. In the upper one the nucleus shows beginning segmentation, but still has the continuous band of chromatin; in the other, segmentation has not begun. These cells are therefore both band metamyelocytes. They are found in normal blood in a percentage of about 4 to 7.

The interpretation of the maturity of the neutrophils constitutes the essential part of the Schilling index. In the differential count the other elements of the white blood cell picture are important. The significance of the number of monocytes, lymphocytes and eosinophils is familiar to those interested in hematology. In the acute otologic infections discussed in this paper, as in most severe infections, the monocytes and lymphocytes are markedly decreased and the eosinophils

absent. The return of increased numbers of monocytes and then of lymphocytes, and finally the reappearance of eosinophils herald convalescence, as indicated by a blood picture that is returning to normal. The more unusual types of cells, the myeloblasts, lymphoblasts, macropolycytes, etc., have their significance too, but a discussion of these is not within the scope of this paper.

The technic of preparing the blood stains in computing the Schilling index is the same as that for the ordinary differential count, using the cover glass method and Wright's stain, which is simpler and requires less time than the Giemsa stain.

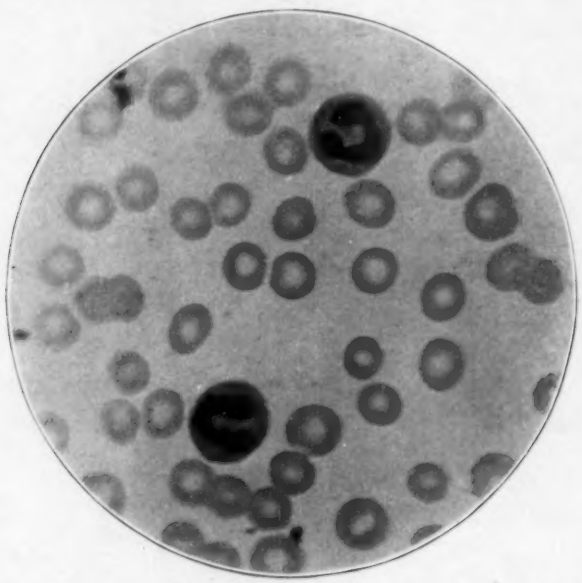

Fig. 2.—Most common forms of immature neutrophils.

Several investigators have recently applied the Schilling index to a variety of infectious diseases. Among recent publications in this country are the articles of Weiss [1] and Reznikoff.[2] It seems to be the consensus that the differential interpretation of the white blood picture according to the Schilling method gives reliable information as to the presence and intensity of an infectious toxemia. When performed at regular intervals, it indicates the prognosis, at times giving this information before any other signs are available.

1. Weiss, A.: Newer Hematologic Aspects of the Neutrophils in Infections, Am. J. M. Sc. **174**:45, 1927.

2. Reznikoff, P.: Immature White Blood Cell Counts in Infectious Diseases, J. A. M. A. **93**:963 (Sept. 28) 1929.

The studies that form the basis of this paper were undertaken at the suggestion of Dr. Harris P. Mosher and Dr. Philip E. Meltzer to determine the practical value of the Schilling index in otolaryngologic pathology. It is apparent from what has been said that these examinations are related to acute febrile infections. Since the proportion of infections of the ear producing a toxemia is so much greater than that of toxic states arising from the other types of cases, this study has been concerned chiefly with otology. A total of eighty cases has been studied, sixty-five of which were in patients admitted to the ear service of the Infirmary who were suffering from one or more of the major complications of otitis media.

Simple mastoiditis presents a varying picture clinically dependent on several factors. In the majority of cases, however, the diagnosis offers no grave problem to the otologist. But the patient frequently seen on a busy otologic service who manifests marked systemic reaction to an infection that evidently began as otitis media presents a different problem. It becomes imperative then to estimate the intensity of the infection and to determine whether or not this pathologic process has progressed beyond the extent of simple mastoiditis. It is in these cases that the Schilling index is especially helpful. Often the total leukocyte count and the ordinary differential estimation do not give reliable information.

A well walled off infection, reflected in the patient by the absence of fever or by only a slight febrile reaction, occasionally gives a normal number of immature neutrophils. Children, who are generally more resilient than adults, may have more marked evidence of infection but show only slight elevation of the immature count, approximately from 8 to 10 per cent. When the count has gone above 12 per cent, however, in cases of acute mastoiditis, surgical intervention is inevitable. In over one fourth of the cases of otologic infection studied, clinical evidence of severe toxemia was present. One or more of the following conditions was present: lateral sinus phlebitis or thrombosis, septicemia, perisinus abscess, extradural abscess, infection of the meninges and invasion of the brain.

In the meningeal complications, the immature count varied a great deal, depending on the presence of extensive disease of the mastoid or complicating sinus phlebitis, etc. In four cases with the history of recent otitis media followed by the sudden onset of signs of complication suggestive of meningeal involvement, the immature count was markedly elevated only as a terminal event. In these cases, the condition of the mastoid was not significant evidence that the infection had "leaked"

through. The original infection had been relatively unimportant until it became the apparent focus when signs of complication set in. The absence of a high immature count in the presence of evidence of an early meningitis, as shown by observations on the spinal fluid, rules against the presence of involvement of the lateral sinus, and extensive disease of the mastoid or a collection of pus, even though the temperature may at intervals be rather high.

Before presenting case reports, I feel that it is helpful in connection with the subject under consideration to refer briefly to certain investigations of the leukocyte count under functional conditions. Garrey[3] found that at absolute rest in the recumbent position a normal person has a basal leukocyte count approximating from 5,000 to 6,000 cells, and in an upright position from 8,000 to 10,000 cells. The performance of certain exercises, such as rising from the recumbent to the standing position, walking, the effect of emotional stimuli and the more strenuous physical exertion of running, in from fifteen seconds to two minutes produced a total white cell count that was from three to four times the basal cell count in the milder exertions from and as high as 35,000 in the more severe physical activity. Sabin and her co-workers,[4] in addition to determining that the normal leukocyte level varies in normal persons engaged in the ordinary activity of working about a laboratory, in the ratio 1:2 within the same hour, found that differential counts showed an average neutrophilic leukocyte percentage with a range of from 56 to 78 per cent. Lymphocyte variations were in the proportion of 1 : 3.

The following case reports are offered as representative of the practical value of the Schilling index. Comparison is made between the total leukocyte and the immature neutrophil counts. The figures for the total neutrophil count in the ordinary differential classification are not included. If this figure is determined by adding the mature and immature forms and the curves are plotted, an even greater variability will be evident than is shown in the curve of the leukocyte count in the accompanying charts. In the cases reported, only the pertinent facts are included. The routine laboratory procedures are omitted, unless of importance. Four hour temperature charts were kept, but only the morning and evening records are charted, which show the general trend of the febrile manifestation. The blood counts were made at the same hour of the day as far as possible.

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3. Garrey: Basal Leukocyte Count and Physiological Leukocytosis, Proc. Staff Meet., Mayo Clin. **4**:157 (May 15) 1929.

4. Sabin, F. R.; Cunningham, R. S.; Doan, C. A., and Kindwall, J. A.: Bull. Johns Hopkins Hosp. **37**:14, 1925.

REPORT OF CASES

CASE 1.—R. T., a nurse, aged 22, was admitted to the hospital on Feb. 4, 1930, because of pain in her right ear for four days with postaural tenderness and swelling for two days. These symptoms had followed a cold in the head and a sore throat that had appeared nine days previously. A paracentesis had been performed three days before admission, and a serous discharge had followed. The pain had been only partially relieved by the paracentesis; on the morning of admission, the earache, which had been intermittent, became continuous and gradually increased in severity. She had been febrile for four days, with some sweating but no chills. She complained of tinnitus and deafness in the affected ear. There was no history of headaches, dizziness, nausea or vomiting.

Local examination revealed a profuse seropurulent discharge with a red, thickened membrana tympani and slight sagging of the posterosuperior canal wall. There was no postaural swelling or redness, but exquisite tenderness was elicited on pressure over the mastoid tip. There was pus in the middle meatuses of the nose, and tenderness was present on pressure over the right maxillary sinus. Roentgen examination indicated diffuse thickening but no destruction in the right mastoid and pus in the maxillary and ethmoid sinuses.

On the patient's admission to the hospital (fig. 3, *I*) the temperature was 99.2 F.; the leukocytes numbered 13,000, and the percentage of immature neutrophils was 14. The patient was under observation in bed for the next five days, during which time treatment consisted of nasal and aural irrigations and an ephedrine spray for the nose. During this period the temperature increased, the leukocyte count reached 20,000, and the percentage of immature neutrophils rose to 18.

A simple mastoidectomy on the right side was performed on February 19 by Dr. C. I. Johnson (fig. 3, *II*). The cells were found filled with a mucopus, but the cellular partitions were intact. The day after operation the leukocyte count rose to 36,000, the temperature was lower than it had been the day previously, and the immature cell count was 19. For several days following, the patient showed progressive improvement. The postaural wound was boggy and swollen and discharged profusely.

On February 27 (fig. 3, *III*) the temperature began to rise again, as did the immature cell count which had decreased to 11 per cent. The total leukocyte count, however, was only 11,000, the lowest point during the illness; it remained the same for several days, while the temperature rose, the immature neutrophil count reached its high point of 22 per cent and the patient manifested an increase in the intensity of the toxemia. A blood culture taken on March 1 gave a late growth of a streptococcus which on subcultures proved to be neither a hemolyticus nor a viridans type. Profuse sweating accompanied the rise in temperature, but no significant chills occurred. The sinuses and chest were examined roentgenologically again on March 3; both were normal.

As soon as the blood culture was reported to be positive (fig. 3, *IV*), the right internal jugular vein was ligated and the mastoid wound reopened. The lateral sinus was exposed and incised with free bleeding from both ends. The appearance of the lateral sinus wall was not particularly significant; there was one small area in which it appeared to be slightly thickened. A blood culture taken at this time gave no growth. The immature neutrophil count on the day of the ligation was 16 per cent. The next day it rose to 19 per cent; the temperature, which had come down to normal, rose again. A blood culture taken the next morning returned a growth, at sixty hours, of the same organism found in the first blood culture.

The patient was given a transfusion of whole blood (fig. 3, *V*). Several hours later the immature cell count was down to 12 per cent. For the next four days the temperature remained high, but was not the typical swinging temperature of infection of the blood stream; it had not been this during the previous high febrile periods. During the latter period of complication, the total leukocyte count had ranged between 13,000 and 16,000, which was hardly indicative of the severity of the infectious process. The wound at the site of the ligation became infected and pus was drained from this, following which the temperature came down to normal, the immature count likewise decreased and actual convalescence set in. The total leukocyte count showed little change for several days, remaining around 16,000. When the temperature had been normal for five days and the percentage of immature neutrophils was normal, the final count was 11,000.

Comment.—This case showed a leukocyte count at variance with what would ordinarily be expected in the presence of the clinical signs

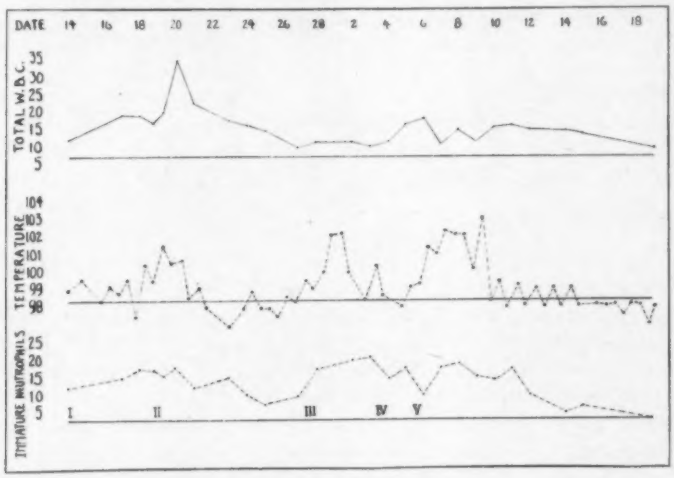

Fig. 3.—Temperature, total leukocyte and immature neutrophil curves in case 1.

manifested by the patient. Following mastoidectomy, when there was evidence of improvement, the leukocyte count rose to its high point, a number double that preceding operation. Then, several days later, when evidence of a lateral sinus phlebitis and invasion of the blood stream was present, the leukocyte count was at a low point, around 12,000.

The immature neutrophil count followed the trend of the toxemia, as evidenced by the clinical signs from day to day.

Case 2.—J. L., a man, aged 22, was admitted to the hospital on Oct. 28, 1929, with a history of pain in the right ear for five days. Ten years previously earache and discharge had developed in the same ear; these symptoms had been present on occasions ever since. Two days previous to admission a paracentesis had been performed with relief from the pain for a few hours. A second paracentesis

was performed the day before admission, with temporary relief. He gave a history of headache, fever, tinnitus and deafness for five days, with postaural tenderness, sweats and chills for two days prior to admission. He had had no dizziness, nausea or vomiting.

Local examination revealed a scant serosanguineous discharge and a thickened, gray membrana tympani. Tenderness could be elicited on pressure over the tip and lower third of the mastoid process. There was no postaural swelling. Roentgen examination showed a sclerosed right mastoid with active infection in the antrum; the left mastoid was sclerosed. The general physical observations were negative. An eye service consultant reported blurring of both disks at the margins with venous engorgement but no measurable choking. The spinal fluid contained sixty-five cells, 75 per cent of which were polymorphonuclears. No organisms were found in a culture of the spinal fluid. A blood culture returned a growth of a staphylococcus which, in the light of subsequent events, seems to have been a contamination.

The temperature on admission (fig. 4, *I*) was 102.2 F.; the total leukocytes numbered 26,000 and the immature neutrophils 30 per cent. A simple mastoidectomy was performed by Dr. H. P. Cahill a few hours after admission. On removal of the mastoid cortex, foul pus escaped. The mastoid cavity was dissected out of sclerotic bone; a soft area was found near the antrum and also near the tip. The bone over the antrum was soft. The dura over the tegmen antri was uncovered, allowing the escape of extradural pus. The lateral sinus plate was removed, revealing a perisinuous abscess of foul pus. The sinus was uncovered well toward the bulb and the posterior margin. The sinus wall was covered with gray necrotic granulations, but seemed thin and compressible.

The temperature, which had risen to 103.8 F., at the time of operation, was between 101 and 102 F. on the following day (fig. 4, *II*); the total white cell count was 28,000 and the immature neutrophil count 34 per cent. The patient's condition seemed worse. He was drowsy but was aroused easily; he attempted to answer questions but was disoriented, could not tell where he was, could not name common articles and complained of the light hurting his eyes. His neck was slightly stiff. A questionable Kernig reflex was obtained; positive Babinski and Oppenheim reflexes were elicited on the left. His pulse ranged between 54 and 60. He complained of frontal headache, and vomited once. The spinal fluid contained 108 cells, 90 per cent of which were polymorphonuclears. No organisms were obtained in a spinal fluid culture. The initial spinal fluid pressure was 108. Consultants from the eye service and from the neurologic service (Massachusetts General Hospital) brought out no additional significant observations.

Because of the report on the blood culture the next day (fig. 4, *III*), together with the observations at operation and the patient's continued critical illness, the right internal jugular vein was ligated. That day and on the three days following, the clinical picture remained subjectively much the same. The neurologic signs persisted. However, continued study by the consultants brought out no new features. The temperature gradually came down to normal, and the immature neutrophil count decreased. The leukocyte count came down to 12,000 while the patient still showed considerable evidence of toxemia; then it rose to 18,000 but gradually declined. When the temperature had been normal for five days and the immature count was normal, the leukocyte count was 12,000.

Comment.—The curves of the total leukocyte and the immature cell counts in this case parallel each other in a general way. The marked

drop in the number of leukocytes following the mastoidectomy is, however, somewhat misleading; it does not reflect the intensity of the toxemia during the immediate postoperative period.

Case 3.—A. B., a woman, aged 22, was admitted to the Massachusetts General Hospital on March 11, 1930, with the chief complaint of grip for three weeks and a discharge from the left ear for one week. A week after the onset of her illness she had called the family physician, who had found the patient febrile and had confined her to bed at home under his care. Her temperature had been "around 102 F. most of the time" before admission. An earache developed fifteen days before the patient came to the hospital; spontaneous discharge resulted eight days later. During the period of illness, she had been nauseated a great deal and had

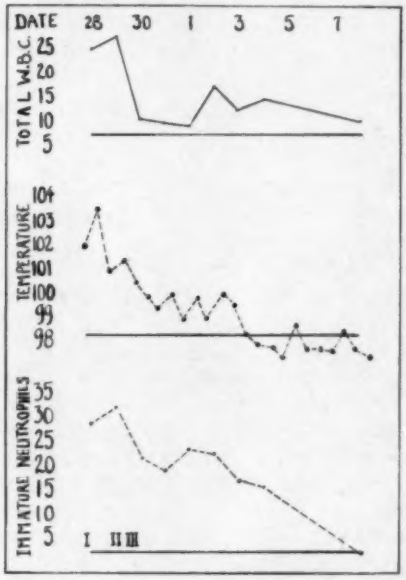

Fig. 4.—Temperature, total leukocyte and immature neutrophil curves in case 2.

vomited frequently. Her past history contained the statement that she had been delivered of twins five months previously and had "not been really well since."

A blood culture taken on the day of admission to the hospital was reported negative.

Early on the morning of March 12, she had a chill lasting from thirty to forty minutes. At 2:30 a. m., she was found staring open-eyed at the ceiling, with immobile facies and fixed jaws. The arms and legs were fixed in flexion; they could be extended against resistance. There was no evidence of a stiff neck or a Kernig reflex. The fundi appeared normal. The patient slowly improved, but her pulse seemed thready; the blood pressure was 63 systolic and 30 diastolic, and a scratchy pericardial friction rub could be heard. A white blood cell count showed 30,000 leukocytes; the red cell count was 3,080,000, and she had a 55 per cent hemoglobin. At 12:30 p. m. she was given a transfusion of whole blood.

following which her condition showed marked improvement. The blood pressure afterward was 90 systolic and 60 diastolic; the pericardial rub was gone, and the patient was rational. On these two days the urine had shown a marked hemoglobinuria.

The patient was seen in consultation that night by an otologist and transferred to the Infirmary. The local examination revealed a thick purulent discharge from the left ear, a red, thickened membrana tympani, moderate sagging of the posterior superior canal wall and periosteal thickening and tenderness over the mastoid tip. The patient seemed weak and toxic.

Roentgen examination revealed a large left mastoid of the fine cell type with marked thickening but no destruction. The right mastoid appeared normal. The sinuses appeared normal in roentgenograms.

Several hours later (fig. 5, *I*) a simple mastoidectomy was performed on the left side by Dr. Philip Mysel. The cortex was thick; on chiseling through it hemorrhagic cells were found. There was a collection of pus under the tegmen antri, which was intact and apparently in good condition. The lateral sinus was exposed down to the knee; it appeared normal. The left internal jugular vein was ligated. When the lateral sinus was opened, free bleeding occurred.

On the day of admission to the Infirmary the temperature had swung from 96.8 to 105.8 F. (rectal). The total leukocyte count on the evening of admission was 30,000 and the immature neutrophilic percentage 32. Several hours following the operation, the leukocyte count was 23,000 and the immature count 23 per cent. The next day, March 14, the patient seemed improved. The red blood cell count was 2,670,000, with 75 per cent hemoglobin.

A transfusion of whole blood was given on March 15 (fig. 5, *II*). The leukocyte count had dropped on the second postoperative day, March 14, to 16,000; the next day it rose to 21,000, and was 18,000 on March 16. The immature neutrophil count during this period had shown a steady decline, reaching a low point of 12 per cent following the transfusion. The wound in the neck became infected. A staphylococcus grew on a blood culture taken on March 16, From that point the total leukocyte count began a steady rise to reach a high point of 49,000 six days later. In contrast with this, the immature neutrophil count, which had risen on March 17 to 16 per cent, was lower on March 18 as the wound in the neck drained; it reached its lowest point of 10 per cent on the following day. It rose sharply with the advent of erysipelas to decrease a little with another transfusion and then rise again with the occurrence of new complications.

On March 18 (fig. 5, *III*) the red blood cell count was 2,420,000 with 62 per cent hemoglobin. Another transfusion was given. A staphylococcus grew on a blood culture taken that day.

Erysipelas developed in the aural wound on March 19 (fig. 5, *IV*). Ten cubic centimeters of serum was given. The red cell count on that day was 3,560,000, with 58 per cent hemoglobin.

Another transfusion of whole blood was given on March 21 (fig. 5, *V*). The red cell count was reported to be 3,560,000 with 50 per cent hemoglobin. A medical consultant made a tentative diagnosis of bronchopneumonia in the lower lobe of the right lung, with metastatic arthritis in the right knee. On daily examination the urine contained large numbers of red and white blood cells and red blood cell casts.

On March 22 (fig. 5, *VI*) the observations in the chest were consistent with the presence of fluid. Roentgen examination indicated fluid at the bases of both lungs and consolidation at the base of the right lung. The red cell count was

2,850,000, with 58 per cent hemoglobin. A blood culture was negative. The patient was transferred to the Massachusetts General Hospital, where trocar thoracotomy was performed and pus was obtained.

On March 24 (fig. 5, *VII*) another transfusion of whole blood was given, with no apparent relief. Death occurred on March 26. Autopsy showed a bilateral empyema, generalized peritonitis and septic pelvic phlebitis. The mastoid wound was partially healed.

Comment.—This case presents an interesting contrast in the curves of the total leukocyte and immature neutrophil counts. The 30,000 leukocyte count and the 31 per cent immature neutrophils on admis-

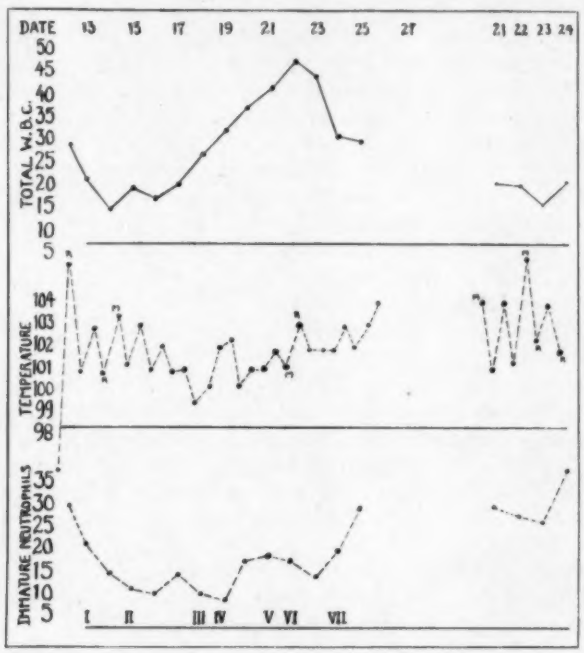

Fig. 5.—At left side of chart, temperature, total leukocyte and immature neutrophil curves for case 3; at right, for case 4.

sion reflected the severe toxemia. The trend of the leukocyte count, from the third postoperative day over the six day period following, to a high point of 49,000 might be interpreted as an indication of a gradual increase in the intensity of the infection, with a resistance that was "holding its own." During this period, however, there were times when the patient seemed much improved clinically, especially following the transfusions. The immature neutrophil count showed a variation with the occurrence of each new event, decreasing after transfusion and rising to the stimulus of each new complication.

The downward trend in the curve of the total leukocyte count during the last four days of the illness might be interpreted as an indication of a "break" in resistance; subsequent events supported this.

However, the curve of the immature neutrophils seemed to indicate more accurately the toxic course during the illness, enabling one to gage more certainly the balance between the infection and the patient's power of resistance to it.

Case 4.—H. F., a woman, aged 32, was admitted to the hospital on March 20, 1930, with a history of earache and pain in the left side of the face for three weeks. She had had a cold in the head with nasal obstruction and a yellow discharge, the onset occurring five weeks before admission. Spontaneous discharge had occurred from the left ear a day after the onset of earache. An otologist had performed a paracentesis for better drainage two days later. Intermittent earache and pain in the left side of her face had continued until three days before she came to the hospital. The day before this pain had abated, she had experienced severe frontal headache, chills for from fifteen to thirty minutes and sweating. Since then she had vomited once or twice daily. The right eyelids began to swell on the day of admission, and she complained of pain "behind the eyes," most marked on the right side.

Local examination revealed a fulness of the membrana tympani which was red and thickened and showed a posterior bulging. The processus brevis could be made out. There was no aural discharge or mastoid signs. The nasal septum was deviated to the right touching the middle turbinate, but there was no secretion in either naris. Moderate tenderness could be elicited on pressure over the right frontal sinus. There was a slight ptosis of the right eyelid, and congestion and chemosis of the right bulbar conjunctiva; the eye was tender to palpation. The left eye showed some congestion of the bulbar conjunctiva.

Roentgen examination indicated a markedly thickened left mastoid, which was of the large, fine cell type, but no destruction. The left mastoid was normal and the sinuses clear.

On admission, the patient's temperature was 104.2 F., and the total leukocyte count 14,200; no differential count was made that evening. It was felt that the more important local process was related to the eyes. A blood culture taken on admission gave no growth.

The next morning a consultant in the eye service made a diagnosis of metastatic uveitis of the right eye, with early similar involvement of the left. The opinion was given that a panophthalmitis would probably result. The temperature that day swung between 101.2 and 104.2 F.; the total leukocyte count was 22,000 and the percentage of immature neutrophils 31.

On March 22, the spinal fluid contained 895 cells with 94 per cent polymorphonuclears but was under normal pressure. The culture of the spinal fluid was reported to contain streptococci. The neck was slightly stiff, and a questionable Kernig reflex was obtained.

Subsequent consultation with the medical and neurologic services brought out no new significant features. On March 23 the spinal fluid showed a slight increase in cells and in pressure, but was negative on culture. Panophthalmitis developed in the right eye, with marked exophthalmos. Cavernous sinus thrombo-

sis seemed the probable diagnosis. Blood cultures taken on March 22 and 23 were negative. Death occurred on March 24. Permission for autopsy could not be obtained.

Comment.—Except for a slight drop in the total leukocyte count at one point, it remained at the same level, about 23,000, for the four days before death. This count was not indicative of the intensity of her infection. The immature neutrophil count, 32 per cent on admission, indicated the severity of the toxemia. It rose to 39 per cent several hours before death.

CASE 5.—E. R., a girl, aged 6, was admitted to the hospital on April 11, 1930, with a history of an earache five days previously, following a sore throat. A paracentesis had been performed soon after the onset of the earache, with relief from pain; a moderate amount of discharge followed. For the entire week prior to admission, she had been feverish, with the temperature "up to 104 F. at times." For the last four days she had complained of nausea and had vomited several times. There was no history of headache, dizziness, sweats or chills.

Local examination revealed a red, thickened membrana tympani in the right ear, with a moderate amount of mucopurulent discharge from a paracentesis incision. There were no mastoid signs. The left ear was normal. The patient appeared to be acutely ill. Roentgen examination indicated a diffuse thickening of the right mastoid with destruction of cells; the left mastoid was normal.

On the morning following admission (fig. 6, *I*), a simple mastoidectomy was performed on the right side by Dr. G. B. Fred. A thick sclerotic cortex was found; within the mastoid were broken-down cells and a large quantity of pus. There was a large perisinuous abscess, and the lateral sinus wall was ashen gray, collapsed and firm to palpation. A large area of the sinus and the cerebellar dura was exposed. On opening the lateral sinus no bleeding occurred until a clot was dislodged from above. The thrombus in the lower end was not disturbed. A small area of tegmental dura was exposed, which appeared normal. The right internal jugular vein was ligated.

A blood culture taken on the patient's admission to the hospital returned a growth of a staphylococcus, but though a subsequent blood culture was negative, the ensuing complications supported the theory of an infection of the blood stream. On the patient's admission the temperature was 105.2 F. (rectal), the total leukocyte count was 7,700 and a differential study showed an immature neutrophil percentage of 32. The spinal fluid contained 31 lymphocytes and was under normal pressure; no organisms were found on culture.

A blood culture on April 13 was negative. The red blood cell count was 3,003,000, with 70 per cent hemoglobin.

On April 15 (fig. 6, *II*) a transfusion of whole blood was given. On April 16 (fig. 6, *III*) a painful swollen right ankle developed. The patient was transferred to the orthopedic service of the Massachusetts General Hospital where an abscess was incised and pus obtained. Following this the temperature dropped; the immature neutrophil count, which had risen slightly in its descent when the arthritic condition appeared, came down to 7 per cent. It rose again during the next three days as another abscess became evident at the right sternoclavicular joint; this also was drained. Following a decrease again in the immature neutrophil count, another rise took place. There was an increase in temperature over a four day period, but the patient did not appear to be worse. On April 28, the

postaural wound began to drain more than it had been doing and the temperature then gradually came down to normal with a return of the immature neutrophil count to normal limits.

Comment.—The striking feature of this case is the fact that the total leukocyte count was misleading; except on two occasions, it remained within what is ordinarily assumed to be the limits of a normal count. On the day the first joint complication appeared, it rose to 21,000; there was also a rise from 7,500 to 12,000 on the day the second joint infection appeared. These complications were a minor part of the picture as compared with the pathologic process on entrance.

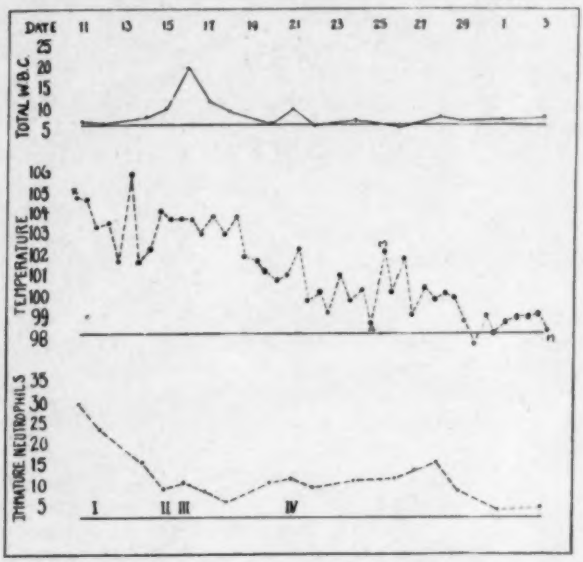

Fig. 6.—Temperature, total leukocyte and immature neutrophil curves for case 5.

The immature neutrophil count, however, accurately reflects the intensity of the toxemia from day to day.

Case 6.—R. M., a boy, aged 9, was admitted to the hospital on Jan. 2, 1930, with the following history: In July, 1929, a simple mastoidectomy was performed on the left side by a surgeon in the city where the patient resided. "The wound never completely healed" and discharge began again about ten days before he came to the Infirmary. This recurrence of discharge was preceded by a cold in the head, slight pain in the ear and frontal headache for two days. For four days previous to admission, he had been febrile; for two days previous to admission he had been nauseated, and had vomited twice the day before, and once on the day of admission. There was no history of chills, but he had had sweats for two days. There was no postaural tenderness or swelling.

Local examination revealed the canal of the left ear filled with thick yellow pus and a red, thickened, boggy membrana tympani. A postaural fistula was discharging pus. The tonsils were hypertrophied and injected; there was injection of the pharynx and a mucoid postnasal discharge. Roentgen examination indicated an absence of cells in the left mastoid and a normal right mastoid.

The additional positive observations were: positive Babinski reflexes and a suspicious Kernig reflex; slight blurring of the disk margins, which was most marked nasally; a clear spinal fluid which contained 200 cells (75 per cent polymorphonuclears) and no organisms; an initial spinal fluid pressure of 180, and normal dynamics in the Tobey-Ayer test. A blood culture taken on admission gave a growth of *Streptococcus hemolyticus*. On admission the temperature was 104.8 F. (rectal), the leukocytes numbered 33,000, and the percentage of immature neutrophils was 31.

A secondary mastoidectomy was performed by Dr. Philip E. Meltzer, several hours after the patient's admission (fig. 7, *I*). The bone in the tegmental area was found to be very soft and was removed, exposing an area of dura. It appeared congested and thickened. A large portion of the lateral sinus plate below the knee was removed. The sinus covering appeared normal; it was soft and easily compressible.

The postoperative course was eventful. For several days the patient complained a great deal of frontal headache. This was relieved by lumbar puncture. On the first postoperative day the spinal fluid was under a pressure of 130 and contained 190 cells, 68 per cent of which were polymorphonuclears; no organisms were found on culture. The cell count gradually decreased on the succeeding days.

On January 6 (fig. 7, *II*) the left internal jugular vein was ligated. A blood culture on January 7 was again positive for the organism previously obtained. A transfusion of whole blood was given on that day (fig. 7, *III*).

Frequent examination by an eye service consultant revealed an increase in blurring of the disks and in tortuosity of the veins. The neurologic service consultants reported no new observations except that on January 10 the patient seemed to use his left arm better than the right one and the Babinski reflex was obtained on the right side only.

With signs of increasing pressure in the fundi and continuance of headache, the development of a pathologic process in the brain seemed probable. Subsequent examination of the spinal fluid, however, showed a decrease in the number of cells, although the pressure was 260 on January 16. The headaches were relieved by spinal fluid drainage.

On January 16 (fig. 7, *IV*) pain developed in the lower right side of the chest; it was worse on breathing. The pediatric service consultant made a diagnosis of a localized pneumonia, probably embolic in origin. By January 19 the signs and symptoms were less marked; five days later the chest was reported clear.

On January 17 a blood culture again was reported positive for *Streptococcus hemolyticus*. On January 18 the red blood cell count was 3,820,000, with 70 per cent hemoglobin. On January 19 the spinal fluid contained only 6 cells, but the pressure was 310. The spinal fluid protein was 34, the sugar 65 and the chlorides 673.

On January 20 the lateral sinus was reopened down near the bulb, allowing the escape of several drops of thick, creamy pus. Another transfusion of whole blood was given.

On January 24 the lateral sinus was again probed and a small amount of pus escaped. A blood culture taken on January 25 was reported negative. On this

date the spinal fluid was under a pressure of 210, and contained only 2 lymphocytes. The neurologic consultant reported no new features.

On January 27 a blood culture again produced a growth of *Streptococcus hemolyticus*. The red blood cell count was 3,430,000, with 65 per cent hemoglobin.

On January 30 (fig. 7, *V*) the lateral sinus was again explored to find the source of continued trouble. The descending portion below the knee was incised and about 15 minims of pus escaped. A partly necrotic clot escaped on examination at the torcular end; beyond this there was a small amount of fresh looking clot, the removal of which produced fairly free bleeding. The patient received another transfusion. From then on his recovery was uneventful.

Comment.—Blood studies were not made as frequently in this case as in the preceding ones, yet they show the value of the immature neutrophil estimations even when performed at greater intervals.

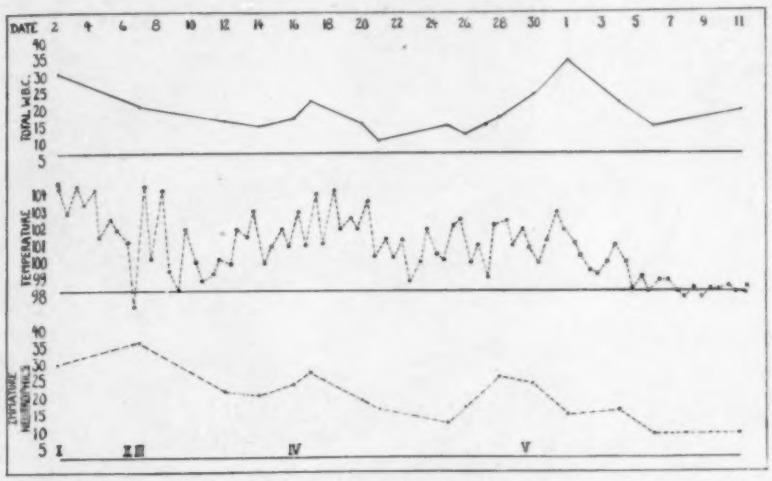

Fig. 7.—Temperature, total leukocyte and immature neutrophil curves for case 6.

The total leukocyte count at various points in the course of the illness is not what would be expected. At the time of admission, it indicated a severe infection, but there followed a period when the count was little above normal limits while the patient showed evidence of severe septicemia. Later in the course, the total leukocyte count was at its high point of 36,000 when there was clinical evidence of improvement and a promising drop in the immature neutrophil count, which marked the definite onset of convalescence. In a comparison of the curves of the two types of white blood cell counts it is apparent that a knowledge of the percentage of immature neutrophils gives an accurate impression of the intensity of the toxic state and information as to prognosis, while the total leukocyte count appears to be misleading at frequent periods in the course of this illness.

SUMMARY

The total leukocyte count and the ordinary differential estimation are not reliable indications in otologic infections of the intensity of an infection or of the resistance of the patient to the toxemia resulting from this infection.

The response of the bone-marrow to the effect of a bacterial toxin results in the presence of immature neutrophilic leukocytes in the blood stream. There results a change in the quality of the neutrophils which are considered to be the active factors in the resisting forces. The percentage of these immature neutrophils reflects the presence and intensity of an infectious process. The numerical relation of these cells from day to day gives an accurate indication of the trend of the toxemia, thus knowledge of the patient's resistance, and light on the question of prognosis.

THE AFTER-EFFECT OF EPINEPHRINE CHLORIDE AND EPHEDRINE HYDROCHLORIDE ON THE MUCOSA OF THE NASAL SEPTUM *

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The symptomatic relief given by epinephrine and ephedrine when applied to inflamed mucous surfaces has caused them to become very popular in the treatment of ordinary acute catarrhal conditions of the nose and sinuses. In the case of epinephrine, unfortunately, after the immediate constrictor effect of the drug wears off, a condition of acute relaxation of the vasculosa occurs, which is probably worse than the original condition. This observation was made by Potts [1] as early as 1906, when the alkaloid epinephrine was first used by otolaryngologists. He reported three cases of chronic turgescence of the nasal mucosa from continuous use of epinephrine during the hay-fever season and warned against its promiscuous use. This after-effect of epinephrine is a familiar one, and accounts, of course, for the greater usage of the ephedrine derivatives which apparently do not produce this undesirable reaction.

The site of action, as well as the modus operandi of epinephrine and ephedrine when used topically, is a greatly debated question. These drugs belong to the sympathomimetic group, so called because their effect simulates that noted in stimulation of the sympathetic nerve. The accepted site of action of epinephrine is believed to be on the myoneural junction,[2] while that of ephedrine has not been determined positively. The after-effect of epinephrine, i. e., the relaxation of the mucous membrane, has been explained on the basis of an after-paralysis of the sympathetic nerve. This, however, is not acceptable, since electrical stimulation of the cervical sympathetic nerve during the dilatation phase of the nasal mucosa that has been previously sprayed with epinephrine causes the mucosa again to constrict, as does a second application of either epinephrine or ephedrine. The purpose of this work

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* From the Departments of Pharmacology and Anatomy, University of Chicago.

1. Potts, B. H.: Danger from the Careless Use of the Alkaloid of the Suprarenal Gland, J. A. M. A. **47**:1188 (Oct. 13) 1906.

2. Sollmann, T.: Laboratory Pharmacology, Philadelphia, W. B. Saunders Company, 1920.

was to determine, if possible, through histologic methods the mode and site of action of epinephrine and ephedrine when they are applied topically to the mucous membrane of the nose.

TECHNIC

EXPERIMENT 1.—One hundred healthy adult rabbits were selected for the test, fifty of which were sprayed with a 2 per cent solution of ephedrine hydrochloride, and fifty with a 1:1,000 solution of epinephrine chloride. Four hours later, these animals were quickly killed with chloroform, their nasal septums were removed in toto and the following technic used in their preparation. The septums were immersed in a freezing solution of 95 per cent alcohol for 30 minutes. At the end of this period, the mucosa was lifted from the septal frame-work and stretched across a cork matting. It was then placed successively in two baths of absolute alcohol at freezing temperature for one hour each, cleared in a solution of isosafrol for twelve hours, and mounted in balsam.

Numerous experiments were carried out under the direction and suggestion of Dr. R. R. Bensley in the anatomy department of the University of Chicago, before the described technic was selected. The fragility of these tissues makes it almost impossible to prepare them according to standard methods in use for preparations of gross tissue. This method leaves the tissue clear, without hemolyzing the blood, giving it the appearance of an injected specimen with all the vessels intact. It is then possible to study these vessels under the ordinary powers of the microscope. The larger vessels, as well as the capillary bed with their connecting constituents, may be observed. If there is free blood in the interstitial tissues, it can be noted.

Figure 1 represents the septal mucosa of a rabbit previously sprayed with ephedrine hydrochloride and prepared according to the technic described.

It will be noted that the walls of the large vessels, as well as those of the capillaries in the background, are rather sharply defined, while only a little blood has escaped into the tissues of the vascular bed, leaving it clear. One must interpret this as an absence of any great injury to the vessel walls of both the large vessels and the capillaries. Contrast this with figure 2, which represents the septal mucosa of a rabbit previously sprayed with epinephrine chloride and prepared according to the described technic. It is apparent that the walls of both the large vessels and the capillaries are blurred and irregular. They do not have the sharpness of detail noted in figure 1. In addition, blood is present in the vascular bed, apparently having escaped from the smaller capillaries. Also, many more vessels are noted in the capillary bed than are seen in figure 1. One must interpret this as being due to a dilatation of these vessels, which makes them become more apparent.

Figure 3 represents the septal mucosa of an unsprayed rabbit. It is in nowise different from that noted in figure 1, i. e., the rabbits sprayed with ephedrine.

Fig. 1.—Septal mucosa four hours after being sprayed with a 1 : 50 solution of ephedrine hydrochloride.

Fig. 2.—Septal mucosa four hours after being sprayed with 1 : 1,000 solution of epinephrine chloride.

Fig. 3.—Normal nasal mucosa

Fig. 4.—Vessel of the nasal mucosa after the use of a 1:50 solution of ephedrine hydrochloride; the contents appear normal; × 790.

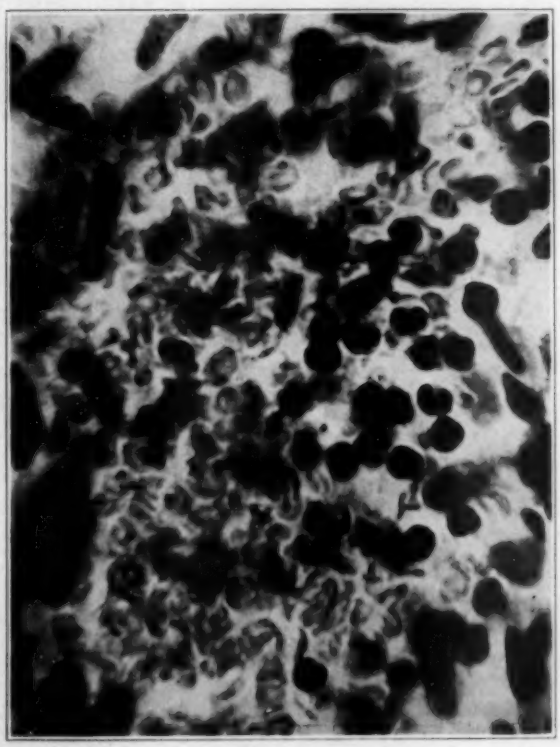

Fig. 5.—Vessel of the nasal mucosa after the use of a 1:1,000 solution of epinephrine chloride. Note the peripheral distribution of white cells and their increased number. They are shown escaping into the tissues; × 790.

Conclusions.—1. A 1 : 1,000 solution of epinephrine chloride sprayed on the nasal mucosa of a rabbit after four hours causes the vessels, which at first are constricted, to dilate and allow blood to escape into the perivascular bed.

2. Ephedrine hydrochloride when sprayed on the nasal mucous membrane, leaves the vessels unchanged after four hours have elapsed.

EXPERIMENT 2.—In order to study this phenomenon further, six animals were sprayed with ephedrine hydrochloride and six with epinephrine chloride. These were killed after four hours and fixed in formaldehyde and Zenker's solution and prepared in the ordinary way for histologic examination. The sections were then cut longitudinally and studied.

Figure 4 represents a vessel of the nasal mucosa of one of the rabbits sprayed with a 1 : 50 solution of ephedrine hydrochloride. It will be noted that only the ordinary number of white cells are present, with the ordinary distribution of these cells as noted in the normal. This may be contrasted with figure 5 which represents a vessel of the nasal mucosa of one of the rabbits sprayed with a 1 : 1,000 solution of epinephrine chloride. The increased number of white cells, their peripheral distribution, and their escape into the tissues is at once apparent. This is the picture of chemotaxis described by pathologists as occurring about areas of inflammation or injury.

Conclusions.—1. A 1 : 50 solution of ephedrine hydrochloride sprayed on the nasal mucous membrane of a rabbit causes no change in the corpuscular content or arrangement of the blood vessels when observed four hours after treatment.

2. A 1 : 1,000 solution of epinephrine chloride sprayed on the mucous membrane of a rabbit's nasal septum causes a rearrangement of the white cells and escape of some cells, from the mucosal vessels not unlike the phenomenon noted in chemotaxis.

SUMMARY

On the basis of these experiments, it is difficult to say whether these changes noted in epinephrine are due to some difference in the physiology of the sympathetic innervation or follows some change in the musculature of the arterioles, capillaries or venules. It seems rather, that epinephrine exerts some direct injury to the vessel walls.

INTRATEMPORAL SUTURE OF THE FACIAL NERVE*

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Anastomosis of the facial nerve was done in 1898 by Faure,[1] who joined the eleventh to the distal end of the paralyzed seventh nerve. In 1903, Korte[2] first did a hypoglossofacial anastomosis. Many others did similar work, and there may have been earlier cases, but they were not published. The objections to these methods have been atrophy of the trapezius muscle and tongue, respectively, plus associated movements of the face and shoulder or the face and tongue. In 1910, Grant[3] endeavored to prevent atrophy by anastomosing the descendens noni to the distal end of the eleventh nerve.

I have not found any reports of intratemporal end-to-end anastomosis of the facial nerve except the one successfully done by Bunnell.[4] Alt gave a method of opening the facial canal, and Ney[5] reported a method of decompression of the nerve and suture worked out on the cadaver. Ney suggested its possible use in Bell's palsy, and further study along this line should be done. He gave a method of protecting the nerve from further compression by utilizing the temporal fascia and muscle to form a tunnel. Bunnell used a piece of the sternomastoid for a similar purpose. Ney stated that most of the lesions of the facial nerve within the canal are probably compressive in nature, and that most of the cases occurring within a few hours or days after operation are probably of this type. I have seen five such cases that developed after radical operation on the mastoid, in all of which the patients recovered within a few months with no treatment except that of loosening the

* Submitted for publication, Oct. 1, 1930.

* From the Department of Otology, University of California.

* Read before the Pacific Coast Oto-Ophthalmological Society, Victoria, B. C., Sept. 4, 1930.

1. Faure, J. L., and Furet, F.: Traitement chirurgical de la paralysie faciale consécutive à un traumatisme intra-rocheux: L'anastomose du facial et de la branche trapézienne du spinal, Gaz. d. hôp. **71**:259, 1898.

2. Korte, W.: Ein Fall von Nervenpropfung: Des Nervus facialis auf den Nervus hypoglossus, Deutsche med. Wchnschr. **29**:293, 1903.

3. Grant, W. W.: Traumatic Facial Paralysis: Anastomosis of Facial Nerve to Spinal Accessory, and the Peripheral End of Accessory to the Descendens Hypoglossi, J. A. M. A. **56**:1438 (Oct. 22) 1910.

4. Bunnell, Sterling: Suture of the Facial Nerve Within the Temporal Bone, Surg. Gynec. Obst. **45**:7 (July) 1927.

5. Ney, K. W.: Facial Paralysis and the Surgical Repair of the Facial Nerve, Laryngoscope **32**:327, 1922.

packing plus massage and electric stimulation. Should this fail, exposure might be in order.

CHOICE OF CASES

It should be stated that only patients with peripheral facial palsies should be operated on. These palsies are complete, whereas the central palsy due to crossed innervation, as well as homolateral innervation of the upper facial division, shows a complete loss of the lower division only.

Those patients who have a complete paralysis on awakening from the anesthesia after operation are the ones on whom operation has been successful. In Bunnell's case and in mine the nerve had been cut about 0.5 cm. below the bend or horizontal semicircular canal. In short, the lesion was distal to the ganglion and caused a loss of taste over the anterior two thirds of one side of the tongue from involvement of the chorda tympani plus a complete facial paralysis. There should be no nerve deafness or labyrinthine symptoms, as these suggest a lesion proximal to the ganglion.

ANATOMY OF THE FACIAL CANAL

Ney quoted MacKenzie, who divided the canal into four parts: vertical, bend or pyramidal, tympanic and labyrinthine. The vertical extends from the bend to the stylomastoid foramen, which is superior, medial and about 7 mm. anterior to the tip of the mastoid. It is about 3 mm. posterior to the meatal posterior surface and is usually separated from the mastoid cells by 1 or 2 mm. of solid bone. In some specimens the cells appeared to communicate directly with the canal.

The bend is the portion in which the direction changes from vertical to horizontal and is from 2 to 6 mm. long. The tympanic segment extends from the bend to the genu, lying between the horizontal canal and the stapes, and proceeds superiorly and slightly anteriorly to the junction of the roof and inner wall, where it forms the genu and leaves the tympanic cavity. The labyrinthine portion extends through the petrous bone, curved toward the cochlea. The diameter of the labyrinthine portion is 1 mm.; that of the genu, from 2.5 to 3 mm., which narrows to 1 or 1.5 mm. in the tympanic region. The average adult canal measures about 25 mm.

SURGICAL INJURY

The vertical and tympanic sections of the canal are the most commonly injured in surgical intervention of the mastoid: the former through searching too low for the antrum, fracture of the tip with rongeurs or chiseling the posterior wall of the canal at too acute an angle in the radical operation, and the latter from breaking off the bridge

too violently with the gouge uncontrolled, or the injudicious use of the curet in the aditus. In many cases of trauma in which the compression or bruising has not been too great, the patients recover, as do those in whom the nerve exposed by caries or cholesteatoma lies bare and is compressed by packing too tightly. They usually do not show a complete paralysis on recovery from anesthesia.

The patients showing a complete functional loss on awakening have had the nerve cut or bruised, usually beyond Nature's repair. Adson [6] said, "If the paralysis occurs immediately after the operation, it may be due to hemorrhage within the canal or to pressure or traction; only occasionally is it due to complete severance of the nerve." This is a comfort to the surgeon, but probably is not correct if it is immediate and complete.

He quoted Lillie as "believing that the facial nerve is most commonly injured in the area on the promontory just anterior to the ampullar end of the horizontal canal." This would be so in radical mastoidectomy and would preclude anastomosis, as the proximal end would probably be too short.

In so-called simple mastoidectomies I believe that the vertical portion is more commonly injured and can be repaired if necessary.

TIME OF REPAIR

After complete healing and asepsis have been reached, several months should elapse to allow spontaneous recovery before suture is attempted. In the presence of sepsis, failure is certain. During this period, massage and electric stimulation should be employed to prevent loss of muscle tone and contractures. If a period longer than a year has elapsed, recovery is doubtful.

REPORT OF CASE

History.—Nellie C., aged 23, the patient of another aurist, was seen on May 2, 1929. She had left facial paralysis on awakening from anesthesia after a simple mastoidectomy eleven months before examination.

She could not remember having had any of the diseases of childhood. Operation had been performed on the mastoid on May 5, 1928. Slight frontal headaches had been present since the operation. There was a slight postnasal drip. There was a slight deafness in the left ear before operation, but at the time of examination that ear was quite deaf. The past history was otherwise irrelevant.

In April, 1928, she had a cold in the head; nine days later spontaneous rupture of the drum of the left ear occurred after four days of pain. For seven days pain persisted in the left ear and over the mastoid. Operation was performed for mastoiditis; the face was paralyzed when the patient awakened. At first she was unable to close the teeth together.

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6. Adson, A. W.: Surgical Treatment of Facial Paralysis, Arch. Otolaryng. 2:217 (Sept.) 1925.

Examination.—On examination, there was left facial paralysis. The pupil of the left eye was slightly smaller than that of the right. She could not close the left eye. The reaction of the pupils was normal. There was no nystagmus, and the disks were normal. There was a scar from mastoidectomy on the left ear; the right arm was normal; the left drum was flattened. The heart and lungs were normal. The blood pressure was 102 systolic and 60 diastolic. The striae of the abdomen were normal. The extremities were normal. The reflexes were normal. Urinalysis gave negative results.

Examination of the blood showed: hemoglobin, 90 per cent; red cells, 4,640,000; white cells, 6,800; polymorphonuclears, 53 per cent; small lymphocytes, 32 per cent; large lymphocytes, 7 per cent; eosinophils, 2 per cent, and monocytes, 6 per cent. The Wassermann reaction was negative.

The first and second cranial nerves were normal. In examining the third nerve, it was noted that the pupil of the right eye was larger than that of the left. The fourth and sixth cranial nerves were normal. The fifth and the vasomotor nerves were normal. Examination of the seventh nerve showed: Subjectively, asymmetry of the face had been present for eleven months; objectively,

Fig. 1.—Appearance of patient before operation: *A*, with the face in repose; *B*, with the patient showing the teeth, and *C*, with the patient closing the eyes.

(1) when the patient smiled, the left corner of the mouth drooped; the right corner drew up; (2) when she opened her mouth, the right side pulled to the right, and the left toward the midline; (3) on wrinkling the nose, the left side was smooth, and the right wrinkled; (4) on wrinkling the forehead, the left side was smooth; (5) when she attempted to close the eyes, the left did not close. The sensation of taste was normal on the right side of the tongue anteriorly; it was absent on the left. Examination of the eighth or cochlear nerve revealed: subjectively, roaring and buzzing in the left ear; objectively, bone conduction was greater than air conduction in the left ear, and air conduction was greater than bone conduction in the right ear. The Weber test was lateralized to the left. The watch was not heard with the left ear, and was heard at 2 inches (5 cm.) with the right ear. The drum of the right ear was normal; that of the left ear was flattened. The vestibular test showed: subjectively, no dizziness and normal gait; objectively, no nystagmus. Examination of the ninth nerve revealed the taste normal on the posterior one third of the left side of the tongue, and absent on the anterior two-thirds. The gag and palate reflexes were normal on the two sides. The tenth, eleventh and twelfth cranial nerves were normal.

The diagnosis was left facial paralysis, peripheral.

Operation.—The old scar was opened and excised, the wound being extended well down past the tip. The facial ridge was removed, and the nerve was found to have been severed at the knee by a small instrument, probably a small curet, in searching for the antrum at too low a point, and a gap of 2 mm. was present. As a result, a knoblike neuroma was present on the proximal segment, and the proximal end of the distal trunk was frayed out. By means of very sharp gouges, the nerve was exposed and lifted out from the vertical canal and stylomastoid foramen; it was also removed from its canal between the oval window and the horizontal semicircular canal by means of a cataract knife. The two ends were removed from the adjacent fragments of nerve, at least 2 mm. from each end. When an attempt was made to bring the two ends together, the vascular silk and very fine needle which were used tore out before approximation occurred. By further freeing of the scar in the neck and the nerve at the stylomastoid foramen, more mobilization resulted, and the nerve was brought together without tension (with vascular silk and needle). When the nerve was freed, the incus was dislodged, and the skin of the posterior canal wall was broken in two places.

Fig. 2.—Appearance of patient after operation: *A*, with the face in repose; *B*, with the patient showing the teeth, and *C*, with the patient closing the eyes.

The posterior wound was closed (clips), except for a space for a small gauze wick; a gauze wick was lightly inserted in the canal.

Course.—The postoperative course was uneventful, except that on the fourth and fifth days the patient was dizzy and vomited; she presented a fine nystagmus to the right, a normal temperature and no rigidity of the neck. The wick in the canal and the clips were removed on the fourth day. The patient was ambulatory on the tenth day and was discharged from the hospital on the eleventh day. No dressing or medication was applied to the mastoid or the middle ear. The patient was instructed to avoid traction on the neck while washing. The slight serous discharge cleared, and the wound epithelized smoothly, the drum falling against and adhering to the promontory and sealing off the eustachian tube. The skin bridges of the posterior canal wall gave way, and the picture was that of a healed wound after a radical operation on the mastoid. The nerve's course under the skin could not be traced.

The nerve was allowed to remain uncovered by muscle flaps, since I had seen no ill effects following a similar complete exposure of the nerve in an attempt to remove a carcinoma of the canal which had surrounded it. The growth was

removed from the nerve, but since it was invading the labyrinth and could not be completely exenterated, the nerve was allowed to remain, lying on the bone, and it recovered after a transient weakness. This gave a lead which I followed in this case and shows, I think, that covering flaps are not essential. The nerve is kept moist by the blood clot and serum and is gradually covered by granulations and epithelium.

Gentle massage and attempts at facial movements were used daily, all traction on the neck being avoided. No return of function was noted for seven months, when the patient noticed that food no longer trickled from the corner of her mouth. One month later she noted that the eye could be almost closed, and her face no longer looked "lopsided."

At the time this paper was written, fourteen months after suture, the face in repose was symmetrical, the nasolabial fold and a dimple had returned, the eye could be completely closed, and there was no difficulty in eating, but the use of the frontalis mucle had not returned. This, I believe, will occur. The defect in taste was, of course, present, since the chorda tympani was not repaired.

Emotional control was good, but there was some lack of voluntary control due perhaps to the crossing of bundles by rotation after the suture. The patient did not show the upper teeth well on command, yet on closing the eye tightly the corner of the mouth drew up perfectly. This movement was gradually improving.

The hearing showed the Weber test slightly lateralized to the right. Air conduction equaled bone conduction; air conduction was normal to all fôrks, and the conversational voice was accurately repeated at 20 feet (650.6 cm.). The tinnitus had disappeared, for which I have no explanation.

CONCLUSION

1. Intratemporal facial anastomosis is possible and gives good emotional control. It should be attempted in complete postoperative facial paralysis, after a suitable time has elapsed for spontaneous recovery.

2. This method may offer hope as a decompressive procedure in cases of Bell's palsy in which the patients fail to recover.

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RÔLE OF THE SYMPATHICUS AND PARASYMPATHICUS IN THE ETIOLOGY OF ATROPHIC RHINITIS*

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In the long years of search for the etiology of the simple and the fetid form of atrophic rhinitis, much work has been done to determine the rôles that the sympathetic and parasympathetic nervous systems play in the production of this condition. Particular stress was laid on the vasoconstrictor and the vasodilator action of these two antagonistic nervous apparatus, and the remainder of the symptoms were explained as a result of the disturbed circulation. Lack of the proper supply of blood would cause atrophy of the bony framework of the nose and the lining mucosa with degeneration of the mucous glands, producing a scant, inspissated secretion with the formation of a crust and fetor.

Zarniko [1] considered atrophic rhinitis as trophoneurosis; his theory was supported by Spiess,[2] who as the first, found that the mucous membrane is hypesthetic in these cases, a phenomenon that is familiar to those who treat many patients with atrophic rhinitis. This theory has been abandoned since the existence of the so-called trophic nerve fibers has not been generally admitted.

In a recent article, Ruskin [3] came to the conclusion that "atrophic rhinitis, when not due to sinusitis, is a disease caused by the abnormal sensitiveness of the vasoconstrictor nerves, which results in a continuous or intermittent spasm of the smaller arteries and arterioles." From a review of the literature on this subject and from my own observations in the conservative and surgical treatment in cases of atrophic rhinitis, I am unable to agree with his conclusions.

Clinical observations, experimental work, the study of the histopathology, surgical therapy and the therapeutic use of certain drugs with selective action on these nerves furnish the bases for the investigation of a possible etiologic importance of a disturbed function of the sympathicus and parasympathicus in atrophic rhinitis.

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* From the Oto-Laryngological Department, Beth Israel Hospital.

1. Zarniko: Die Krankheiten der Nase, ihrer Nebenhöhlen und des Nasenrachenraumes, Berlin, S. Karger, 1910.

2. Spiess, G.: Beitrag zur Ozenatherapie, Ztschr. f. Hals-, Nasen- u. Ohrenh. **4**:273, 1922-1923.

3. Ruskin, S. L.: Atrophic and Vasomotor Rhinitis: The Physiology of the Nasal Mucosa, Arch. Otolaryng. **11**:698 (June) 1930.

A case of a fracture of the basis cranii, in which a hematoma developed in the region of the sphenopalatine ganglion, was reported by Helsmoortel.[4] Two weeks after the accident, there developed, besides an anesthesia in one cheek, an atrophy of the mucous membrane on that side. A similar case of unilateral ozena after an injury to the sphenopalatine ganglion was reported by Rebattu and Proby,[5] and another one, caused by a bullet lodging in the posterior ethmoid, by Belinoff.[6]

Contrary to these reports are the observations made after resection of the sphenopalatine ganglion for neuralgia of the second branch of the fifth nerve. Caldera[7] severed the second branch on a rabbit and inoculated its nose daily for thirteen months with fresh material from human ozena without being able to produce the slightest change in the mucous membrane. Schatz,[8] after resection of the sphenopalatine ganglion in three cases, observed the nasal mucosa for five months without noticing atrophic changes.

Lautenschläger[9] described thickening of the nerve sheaths in the mucosa of the nasal cavity and of the sinuses. In cases that have progressed further, one sees hyaline degeneration of both the sheath and the axis cylinder. Gallotti,[10] in twelve cases of ozena, showed this degeneration along the second branch of the trigeminus, while the first and third branches showed no pathologic changes.

The most exhaustive study on this subject is the recent contribution of Klaus Vogel,[11] who examined the sphenopalatine ganglion histologically in a series of fifty-one cases of general disease with special consideration of atrophic rhinitis. He also removed the ganglion on one side in ten cases in which he performed a bilateral Lautenschläger-Seiffert operation for ozena. This gave him an excellent opportunity to study the influence of the ganglion on the nasal mucosa. He came to the conclusions:

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4. Helsmoortel, J., Jr., and Van Bogaert, L.: Un case d'ozene traumatique unilateral, Rev. d'oto-neuro-opht. **6**:417 (May) 1928.

5. Rebattu, J., and Proby, H.: Ozène experimental, Arch. internat. de laryng. **33**:804 (July-Aug.) 1927.

6. Belinoff, S.: Contribution à l'étude de l'ozène unilatéral d'origine traumatique, Rev. de laryng. **49**:576 (Oct.) 1928.

7. Caldera, C.: Experimentelle Versuche zur Kaninchen-Ozena, Ztschr. f. Hals-, Nasen- u. Ohrenh. **1**:162, 1922.

8. Schatz, K. W.: Welchen Einfluss hat das Ganglion sphenopalatinum auf die Entstehung der Ozena? Ztschr. f. Laryng., Rhin. **17**:312 (Jan.) 1929.

9. Lautenschläger, A.: Rhinitis atrophicans in Denker and Kahler: Handbuch der Hals-, Nasen- u. Ohrenheilkunde, Berlin, Julius Springer, 1928.

10. Gallotti: Arch. ital. di otol. **2**:186, 1920.

11. Vogel, Klaus: Histopathologische Befunde der Ganglion sphenopalatinum mit besonderer Berücksichtigung der atrophischen Rhinitiden, Ztschr. f. Hals-, Nasen- u. Ohrenh. **21**:507 (Feb. 16) 1929.

1. In genuine atrophic rhinitis, an isolated atrophy of the sphenopalatine ganglion occurs, but the Gasserian, geniculate, ciliary and jugular ganglia remain normal. 2. No proof can be furnished that a primary disease of the ganglion is responsible for atrophic rhinitis. This has been found by virtue of histologic examinations of the ganglion in ozena, in acute and chronic inflammations of the nose and nasal accessory sinuses, in infectious and noninfectious general diseases and on the basis of clinical experiences after extirpation of the ganglion. Therefore, the disease of the ganglion must be looked upon as a secondary condition. Another cause, so far unknown, must be accepted for ozena.

By recalling briefly the anatomy of the sphenopalatine ganglion, one knows that the vasoconstrictor fibers of the sympathicus, after they emerge from the upper cervical ganglion, reach the sphenopalatine ganglion through the periarterial plexus of the carotid and the deep petrosal; while the vasodilators, derived from the hindbrain, constitute the nervus intermedius of the facial ganglion and eventually enter the greater superficial petrosal which supplies the ganglion with one of its roots. The latter also carries the secretory fibers to the mucous glands of the nasal mucosa. All of the nasal chamber except its anterior part and part of the ethmoid, which are supplied by the ethmoidal nerve from the first branch of the trigeminus, is innervated by the branches of the sphenopalatine ganglion.

From the classic experiment in physiology, it is known that after extirpation of the superior cervical ganglion in the rabbit, there results an engorgement of the blood vessels of the auricle on the same side, bringing about an hypertrophy. With this experiment as a basis for therapeutic measures in atrophic rhinitis, one should aim to remove only the sympathetic fibers and to save, and if possible to stimulate, the vasodilator and secretory fibers of the parasympathicus. But extirpation and blockage of the ganglion interrupts both the dilators and constrictors and also destroys the sensory branches for the mucosa. It is a well known fact that conjunctivitis and ulcers on the cornea will develop after interruption of the sensory fibers of the ophthalmic nerve, a phenomenon which is explained by the lack of reflectory protection afforded by the normal reflex arc. To deprive completely the nasal mucosa of its sensory branches, which, as previously stated, are lowered in their sensitivity per se, I regard as harmful. I have used galvanization and faradization repeatedly in cases of beginning ozena in order to stimulate the sensory end-fibers in the mucosa which normally elicit the desire of the patients to clean the nose from secretion and crusts, with the result that I was able to retard the outbreak of the fully developed symptoms for years.

Out of these considerations, many surgeons undertook to remove only the sympathicus, by what is known as periarterial sympathectomy of the neck.

Casteran [12] and Bertein [13] reported cases in which this has been done. They noticed improvement for a short period after which the condition returned to the old status; they explain this phenomenon by the impossibility of completely removing all the fibers. Six days after a similar operation, Asteriades and Berjis [14] saw all the symptoms of ozena disappear; but they did not follow their cases long enough to reach a final judgment as to the value of this operation.

The improvement of atrophic rhinitis for a short duration following surgical procedures on the head and neck is common and is to be disregarded as a permanent success. Tonsillectomy and adenoidectomy and the various nasal operations, including those that will ultimately result in an aggravation of the symptoms, are followed by temporary congestion and increased secretion of the nasal mucosa. This occurrence, I consider, is due to the resorption of extravasated blood, similar to the effect of a parenteral protein application, since one can obtain the same effect by injections of the patient's own blood in the turbinates.

The recent view that ozena is a nasal expression of a constitutional disorder, particularly of the glandular structures, has led many authors to subject patients suffering from it to a complete physicochemical examination. Hoople and Rawe [15] observed a downward tendency of the blood pressure and an upward tendency of the pulse rate in a series of eighty cases. The result of the investigation of the autonomic nervous system in general by Wiškowský [16] also showed a definite hypotony of both the orthosymphaticus and parasympathicus in cases of atrophic rhinitis.

Muck [17] observed a decrease in the amount of perspiration on the tip of the nose in patients with ozena and explained it as a local manifestation of a dysfunction of the sudorific fibers of the sympathicus which accompany the external nasal nerve. I have exposed my patients to the Bruening electric headbath but was unable to confirm this observation. The results obtained with the Lautenschläger operation and its

12. Casteran, E.: La sympathicectomie périartérielle comme traitement de l'ozène. Ses resultats, Arch. internat. de laryng. **33**:812 (July-Aug.) 1927.

13. Bertein, P.: Periarterial Sympathectomy in Ozena, Arch. internat. de laryng. **7**:861 (July) 1928.

14. Asteriades and Berjis: Periarterial Sympathectomy as Treatment for Ozena, Clinique, Athens, 1926; quoted by Spetsiosis: Folia oto-laryng. **26**:163, 1926.

15. Hoople, G. D., and Rawe, A. W.: Atrophic Rhinitis: Some Findings in a Series of Eighty Cases, Ann. Otol. Rhin. & Laryng. **36**:144 (March) 1927.

16. Wiškowský, B.: Endocrine and Sympathetic Systems as Factors in Causation of Ozena, Časop. lék. česk. **66**:587 (March 28) 1927.

17. Muck, O.: Beeinträchtigung der Schweissabconderung an der Nasenspitze der Ozaenakranken (Anhidrosis-Hyphidrosis). Ein Beitrag zum Verständnis des Wesens der Ozaena, Ztschr. f. Hals-, Nasen- u. Ohrenh. **19**:421, 1928.

modifications,[18] with or without subsequent injections of cholesterol, cannot be explained on the basis of a primary disorder of the autonomic nervous system. In order to stimulate secretion of the nasal mucosa, the drugs of the pilocarpine [19] group have been used in local application. No definite results, however, have been reported from its use.[20]

SUMMARY

No definite conclusion can be reached that the sympathicus and parasympathicus play an important rôle in the etiology of atrophic rhinitis. General sclerosis of the connective tissue, as seen throughout the mucosa, submucosa and the bony framework of the nose in ozena also involves the blood vessels to the extent that the lumen is reduced to a minimum. It is hard to believe that the sclerosis of the blood vessels is secondary to an increased sympathicotonia; nor can one expect that the latter could have any effect on these rigid walls of the blood vessels. It seems more plausible to assume that the chronic inflammatory process in the nose causes degeneration of the nerve fibers secondarily, which, as previously stated, is confined only to the second branch of the trigeminus and the sphenopalatine ganglion, while the sympathetic system as a whole is in a hypotonic state.

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18. Wachsberger, A.: Endonasal Surgery with the Aid of a New Instrument: Simplified Technic, Arch. Otolaryng. **8**:712 (Dec.) 1928.

19. Lewi, F.: Behandlung der Ozena mit Pilocarpin und Rhodankalium, International Congress of Otolaryngology, Kopenhagen, 1928.

20. Delie: Some Aspects of Ozena, Oto-rhino-laryng. internat. **12**:409 (Oct.) 1928.

RHINOPHYMA

ITS TREATMENT AND COMPLICATIONS *

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The treatment for rhinophyma, in spite of its definite pathologic changes, is far from uniform. This may be accounted for by the fact that rhinophyma as a progressive developing deformity of the skin is usually observed in its early stages by the dermatologist and only later, after the tumor has increased considerably in size, does the patient seek the advice of the surgeon. Hence, two methods of treatment prevail, namely, the conservative dermatologic and the surgical. An adequate treatment for rhinophyma must depend entirely on its anatomico-pathologic structure, which I shall discuss.

PATHOLOGIC ANATOMY

The original condition of the skin is acne rosacea, which slowly develops into rhinophyma, usually invading the inferior and anterior parts of the nose, respecting the free border of the alae nasi and diminishing in size from below upward, rarely extending beyond the glabellar region and the lateral border of the nose. The irregular elevations of the tumor are separated by fissures in which sebaceous masses are accumulated. The nose takes on a more or less violet hue spotted by dilated veins. Regardless of the proportion and aspect of the tumor, the lesions are invariably confined to the soft tissues of the nose, the nasal bones, the cartilage and the nasal mucous membrane remaining intact. The modification of the sebaceous glands plays an important part in the pathologic condition. These glands are greatly increased in number and become abnormally developed in size. The subcutaneous tissue and the dermis are the seat of a chronic inflammatory process, but the epidermis generally remains uninvolved. When fully developed, this condition constitutes a repelling disfigurement, relief from which is eagerly sought for.

The following case report is of interest because it involves the problem of treatment for the severe complications which follow a seemingly harmless but dangerous procedure used in the treatment for rhinophyma. In view of the advanced age of the patient, the reconstruction of the resulting nasal defect is of additional interest.

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REPORT OF CASE

A man, aged 64, was admitted to the City Hospital in February, 1927, for treatment for rhinophyma (fig. 1). The patient insisted on having the deformity treated because of the handicap he encountered in providing a livelihood. The

Fig. 1.—Rhinophyma invading the entire nose.

Fig. 2.—After galvanocauterization of the deformity by a dermatologist.

nasal tumor had progressively developed for ten years prior to the patient's admission to the hospital. After galvanocauterization of the hypertrophic parts of the nose, extensive sloughing set in, followed by necrosis of both alae nasi and the tip of the nose. Figure 2 illustrates the condition of the patient at the time he

Fig. 3.—*A*, the two lateral nasal flaps for the lining of the alae nasi and the outlined flap on the forehead for the covering of the defect are raised and sutured back into place for the purpose of increasing their blood supply. *B*, the tubed temporal flap from the forehead has been sutured into the nasal defect; the wound on the forehead is closed after wide undermining of the surrounding skin.

Fig. 4.—The nose is completely rebuilt after the return of the pedicle to the forehead. The scar on the forehead was eliminated by excision, and the wound was easily closed by surrounding skin.

was referred to me for repair of the resulting deformity. The defect of both alae nasi extended to the cheeks and lateral nasal cartilages. The wound was completely healed, and the quadrangular cartilage with the columella was intact and covered at the free border by a thin scar. The general condition of the patient was satisfactory. The nasal defect was repaired by providing a lining from the scarred nasal skin and a covering by means of a tubed flap from the forehead. In the first stage the forehead and nasal skin flaps were properly outlined and sutured back into place for the purpose of increasing the blood supply (fig. 3*A*). In the second stage, which took place four weeks later, the two lateral nasal flaps were raised and turned down to form the lining of the alae nasi; the skin of the forehead previously outlined was raised and sutured into the nasal wound, and its pedicle was tubed (fig. 3*B*). The defect on the forehead was partially closed after extensive undermining of the surrounding skin, and the remaining uncovered surface was left to heal by epidermization.

The transferred skin flaps healed without any disturbance. Six weeks later the pedicle was detached from the nose, opened and replaced in the temporal region. The scar on the forehead was excised, and the defect was easily closed after further undermining of the surrounding skin. This procedure of repeated excision with wide undermining of the surrounding skin was sufficient to bring about the complete closure of the defect on the forehead with a cosmetic result far superior to that following a successful free skin grafting. The part of the skin flap from which the tubed pedicle was detached served for the rebuilding of the right nostril. In the fourth and final stage, the nasal covering was properly modeled by thinning of the transplanted flap of the forehead and excision of scars (fig. 4). All of the operative procedures were performed under local and block anesthesia with 1 per cent procaine hydrochloride.

COMMENT

Treatment for rhinophyma by galvanocautery should be rejected as unreliable and dangerous. Owing to the fact that in this deformity the underlying structures, such as bones, cartilages and nasal mucous membrane, remain uninvolved, these basic parts of the nasal pyramid should not be interfered with. The extent of destruction of the tissue by application of galvanocautery cannot be accurately estimated, and therefore the possible injury to the nasal cartilages and mucous membrane is inevitably followed by necrosis with resulting defect. Outstanding authorities on radiotherapy agree that the roentgen rays and radium also seem to be of no real service in rhinophyma.[1] This coincides with my own experience in these cases. Sporadic reports to the contrary are far from convincing.[2] Decortication of the hypertrophic masses constitutes the logical treatment for rhinophyma. When performed with a knife or razor, it permits a precise removal of the hypertrophic masses and the preservation of a thin layer of subcutaneous tissue and fibrous membrane

1. MacKee, G. M.: X-Rays and Radium in the Treatment of Diseases of the Skin, Philadelphia, Lea & Febiger, 1927, p. 443.

2. Behjet, H.: Behandlung des Rhinophyma, Dermat. Wchnschr. **88**:129 (Jan.) 1929.

overlying the osteocartilaginous frame. The profuse bleeding accompanying this procedure is of minor importance as it is readily controlled by pressure. If the wounded surface is covered with tinfoil and xeroform gauze, it is completely epidermized in a few weeks. The use of the Thiersch graft is unnecessary, as in most instances the new epidermis provides a satisfactory result. Among other points of interest in the report of this case were the great vitality shown by the skin flaps in the reconstruction of the large nasal defect in a man of 64, and the satisfactory result obtained in the closure of the extensive defect on the forehead by a method of repeated partial excision with wide undermining of the surrounding skin.

1125 Park Avenue.

Clinical Notes

A NEW SYSTEM OF LIGHTING FOR THE NASOPHARYNGOSCOPE

New Operating Attachments for Nasopharyngoscope; Modified Combined Antroscope and Irrigating Cannula *

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The nasopharyngoscope is an adaptation of the observation telescope used by urologists in cystoscopic work. The nasopharyngoscope differs from its parent instrument only in size. This change was necessary to make it conform to the anatomy of the nose. The change in the name of the instrument merely denotes its function, viz., nasopharyngoscope, antroscope, eustachioscope, etc.

Holmes was the first person to adopt the use of such an observation telescope in nasopharyngeal observations. Later, Spielberg brought into use the nasopharyngoscope for exploration of the antrum. His device consists of a straight cannula. When it is reenforced with a stylet, one is able to penetrate the lateral wall of the nose and reach the antrum. When the stylet is removed, the cannula becomes a communicating channel between the antrum and the exterior, and permits the passage of a straight nasopharyngoscope of suitable caliber for inspection of the antrum, or a straight irrigating tube for irrigation of the antrum.

The nasopharyngoscope is a tubular telescope of small caliber (about 10½ French). It is made up of two essential parts: (1) a series of lenses and a prism for the observation of the field of vision and (2) a lighting system to illuminate the field of vision.

The field of vision is the image seen through the eye piece of the nasopharyngoscope. This is brought about by a series of lenses and a prism.

The field of illumination is an artificial illumination of the field of vision brought about by an illuminated electric bulb placed at the farthest end of the nasopharyngoscope.

In figure 1 is shown a schematic cross-section of a nasopharyngoscope chiefly in use. The arrow, *X-X'*, is an object within the field of vision. This object is seen through the eye piece after its rays have passed through a prism, *L*, in the direction of the arrows. *R* is the reflecting surface to direct the rays of the vision toward the eye.

O is an electric bulb having its two contacts, *C* and *C'*. The bulb is always in view and is protected from the exterior by a thin glass casing, *G*. The illuminating rays are directed toward the field of vision for illumination, in the direction of the dotted lines.

I have found this system of lighting far from satisfactory. One can readily see the nasopharyngoscope, being of such small caliber; the glass casing, *G*, is very thin and offers no protection to the lamp, *O*, for the heat of the lamp or

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any excessive manipulation of the instrument will cause the protecting glass to break. The lamp and its contacts, *C-C'*, will become completely exposed to outside elements, resulting in short circuits, flickering of the lamp or electric shocks to the patient and operator.

The new system of lighting that I designed tends to overcome this weakness. It is a hidden lamp which directs its rays of light through a prism, which in turn reflects the rays toward the field of vision.

Figure 2 is a schematic cross-section of a nasopharyngoscope demonstrating the new system of lighting. *O* is an electric bulb hidden from view having its

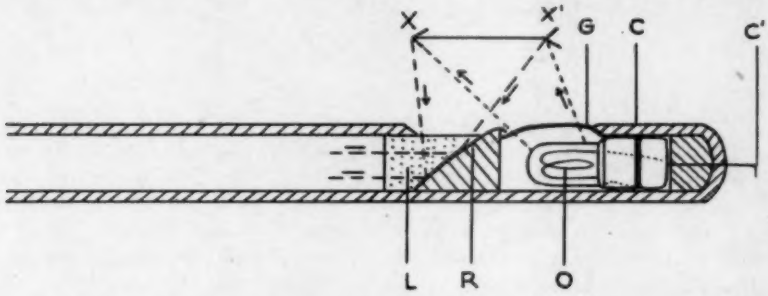


Fig. 1.—Schematic drawing of the old type system of lighting. *x-x'* indicates the object within the field of vision and the field of illumination; *L*, visual prism; *R*, reflecting surface; *O*, lamp; *C-C'*, contacts; *G*, glass casing; dotted lines, direction of the field of illumination; broken lines, direction of the field of vision.

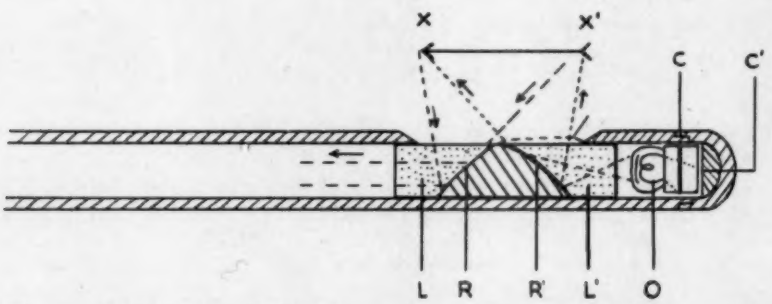


Fig. 2.—Schematic drawing of the new type of system of lighting. *x-x'* indicates the object within the field of vision and the field of illumination; *L*, visual prism; *R*, reflecting surface of the visual prism; *R'*, reflecting surface of the illuminating prism; *L'*, illuminating prism; *O*, electric bulb; *C-C'*, contacts; dotted lines, direction of the field of illumination; broken lines, direction of the field of vision.

two contacts, *C-C'*. The electric bulb may or may not be of a condensing type. The rays of light penetrate the prism, *L'*, until it strikes the reflecting surface, *R'*, then they emerge from the prism, *L*, and fall on the field of vision. The reflecting surface, *R'*, and also the emerging surface of the lens may be plane, concave or convex. This is a matter of choice, depending on whether a diffuse or a concentrated light is required. The reflecting surface, *R'*, must be properly angulated to direct the rays of light on the field of vision.

This system of lighting offers full protection to the lamp and its contacts and also offers a better concentrated light. It may be used in all types of entroscopic instruments.

MODIFIED ANTROSCOPE

The following instrument, which works in conjunction with the nasopharyngoscope, is my modification of the Spielberg antroscope which I previously outlined in this paper. I have modified the Spielberg instrument from a straight trocar to a curved one to permit easier and safer penetration of the lateral wall of the nose. I have also added a side inlet for irrigation and a stopcock to permit a common channel for irrigation and observation.

Figure 4 is a schematic cross-section of the instrument. In figure 4, *I*, the stopcock, *S*, is open to permit the entrance at *B* of stylet *b* for reenforcement of the instrument when penetrating the lateral wall of the nose. It also permits the entrance of the nasopharyngoscope for inspection of the antrum. In figure 4, *II*, the stopcock, *S*, is closed by turning the handle, *H*, about 90 degrees. The instrument is now ready for irrigation of the antrum through the inlet, *I*, into the lumen, *L*, and then into the antrum.

The penetrating portion, *T*, of the instrument is curved away from the long axis of the instrument. It is preferable to have the penetrating portion, *T*, in the same plane as the inlet portion, *I*, but in opposite directions.

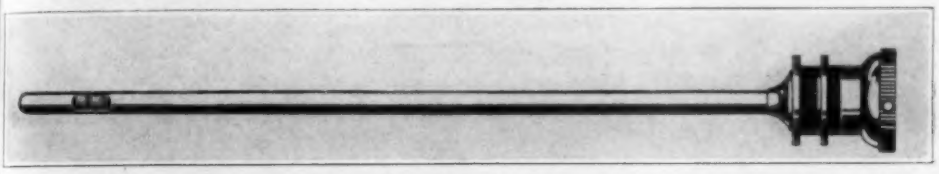


Fig. 3.—Photograph of a nasopharyngoscope with new system of lighting.

CATHETERIZING EUSTACHIOSCOPE

I have already described the next instrument of this series in a previous publication,[1] but I shall repeat the description briefly in order to have a complete series in one paper of all my instruments that work in conjunction with the nasopharyngoscope. I also shall give clearer directions for use, as I failed to do in the previous publication.

The instrument is the catheterizing eustachioscope, which is a combination for catheterizing the eustachian tube under direct vision (fig. 6).

The instrument consists of a nasopharyngoscope and two catheter guides, each of which can be mounted on the nasopharyngoscope with the aid of the sleeve. The guides are marked *R* and *L*.

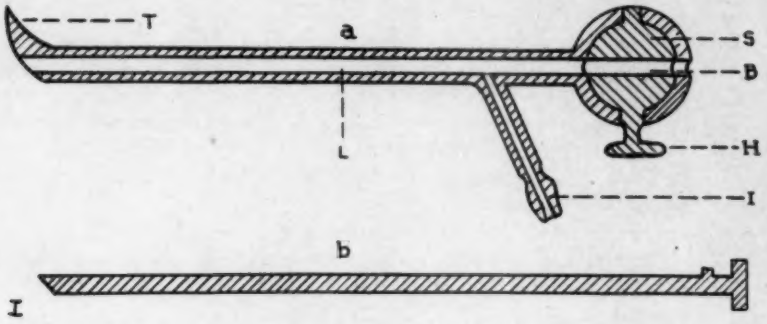
The catheter guide, *R*, is used in combination with the nasopharyngoscope when the right eustachian tube is to be catheterized, and the guide, *L*, in the same manner when the left eustachian tube is to be catheterized.

Direction for Use.—To catheterize the right eustachian tube, the operator holds the eye piece of the nasopharyngoscope directly in front of him with the marking found at the side of the eye piece toward 9 o'clock. He then mounts the catheter guide, *R*, on the nasopharyngoscope. The inlet or handle portion of the catheter guide is also directed toward 9 o'clock. In that position, with the

1. Pitman, L. K.: New York State J. Med. **27**:1061 (Oct. 1) 1927.

patient under slight cocainization, the combined instrument is introduced into the right side of the nose along side the inferior turbinate as far as it will go. In that position the orifice of the eustachian tube will be brought into view. The handle of the catheter guide is turned clockwise toward 10 o'clock, and a suitable catheter, after passing through the lumen of the catheter guide, can easily be directed into the eustachian tube.

To catheterize the left eustachian tube, the procedure is reversed. The operator holds the eye piece of the nasopharyngoscope in front of him with the marking at the side of the eye piece toward 3 o'clock. The catheter guide marked *L* is



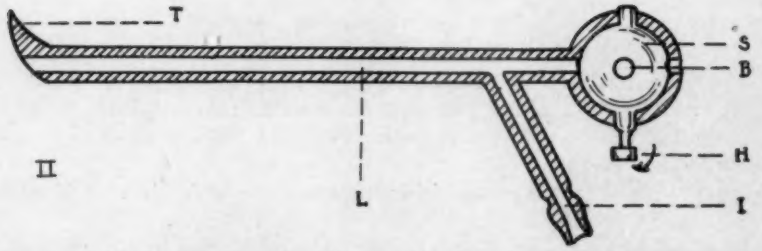


Fig. 4.—Schematic cross-section of a combination trocar and antroscope. In *I*, the stopcock (*S*) is open to receive the stylet (*b*) or antroscope. In *II*, the stopcock (*S*) is closed for irrigation. *T* indicates the penetrating portion; *L*, the lumen; *B*, the inlet for the antroscope; *H*, the handle; *I*, the inlet for irrigation.

mounted on the nasopharyngoscope so that the handle portion of the guide is directed also toward 3 o'clock. In that position the combined instrument is introduced into the left side of the nose along side the inferior turbinate as far as it will go. The handle of the guide is turned counter-clockwise toward 2 o'clock. A suitable catheter can now be directed into the left eustachian tube under direct vision. I have found this instrument adaptable in practically all cases. I have purposely avoided the substitution or the addition of a flap at the terminal end of

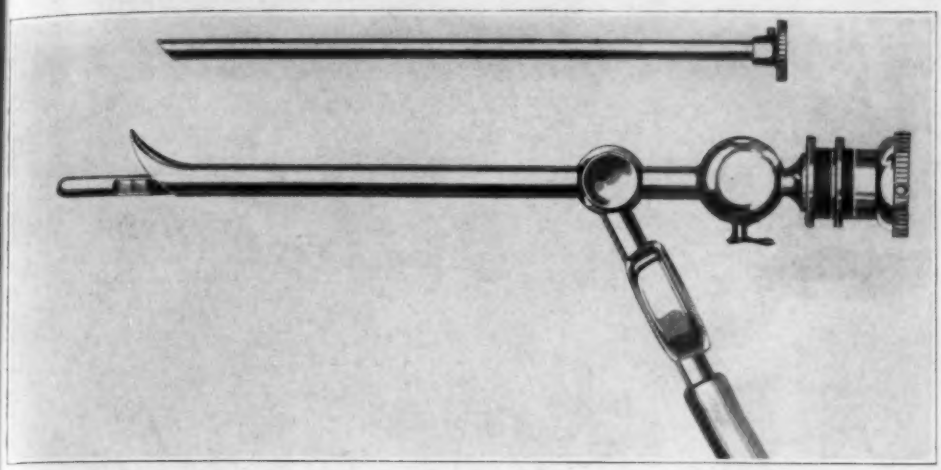


Fig. 5.—Photograph of a trocar with the stopcock open. The nasopharyngoscope is passed through the trocar for inspection.

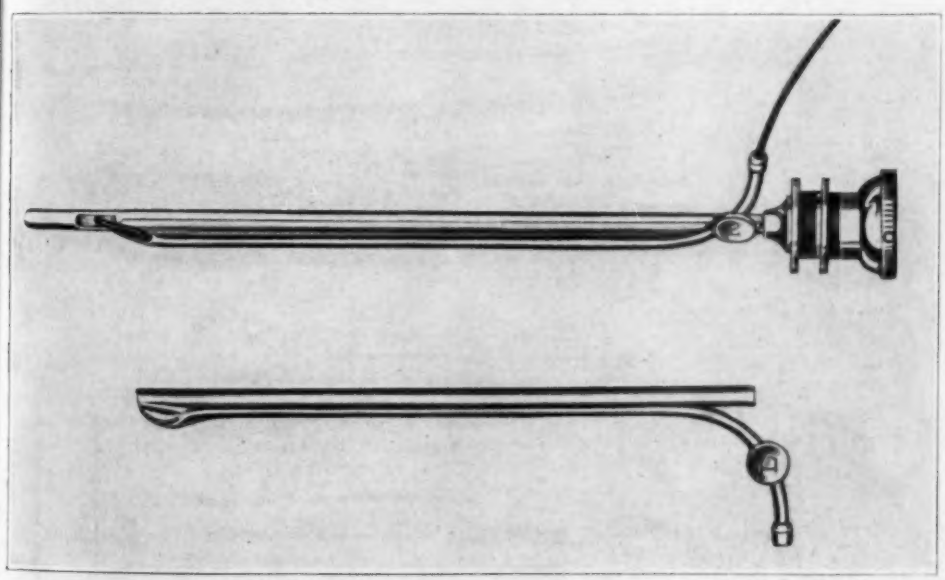


Fig. 6.—Photograph of the catheterizing eustachioscope. The right catheter guide contains the catheter mounted; the left catheter guide is separate.

the catheter guide to guide the catheter. Such an addition or substitution is not only unnecessary, but will complicate a simple instrument. The attachments outlined are simple to operate, inexpensive and the instrument has no unnecessary parts.

SELF-ANGULATING SNARE

A new self-angulating snare for the removal of hypertrophied posterior tips of the inferior turbinate or growths within the nasopharynx is the last of this series of instruments that I have designed.

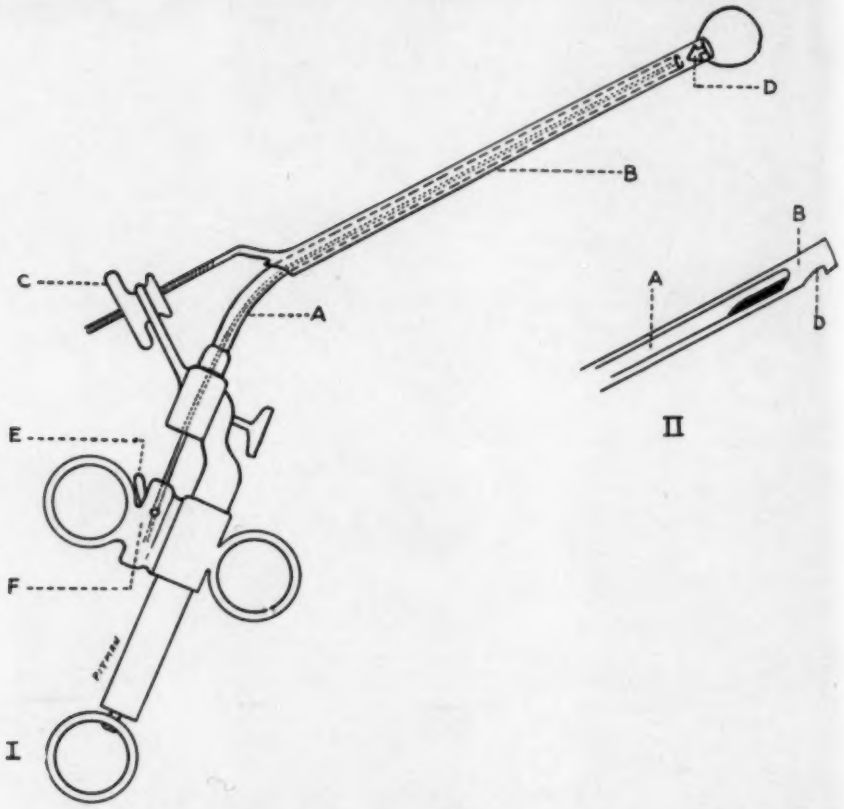


Fig. 7.—Schematic drawing of an angulating snare. *II* is a cross-section of the two cannulas, *A-B*, but at right angles to *I*. *A* indicates the inner cannula; *B*, the outer cannula; *D*, the fenestral opening; *C*, the screw; *E*, the clamp, and *F* the constricting bar.

This type of snare enables the operator by outside manipulation to angulate the loop of wire at various degrees in order to adjust it properly to the base of the growth before snaring. By the addition of a sleeve, properly mounted on the cannula portion of the snare, these growths may be removed under direct vision with the aid of the nasopharyngoscope. However, in removing the hypertrophied tip of the inferior turbinate, the growth being in a constant position, a nasopharyngoscope is not necessary. Therefore a snare without a sleeve is used.

A flap or a bend at the end of the catheter guide will angulate a catheter or a bougie, but such means will not angulate a wire loop. To accomplish this purpose, I have designed a snare having an outer and an inner cannula.

Figure 7, *II*, is a cross-section of the two cannulas, *A-B*., but at right angles to figure 7, *I*. In figure 7, *I*, the outer cannula, *A*, can be slid forward and backward

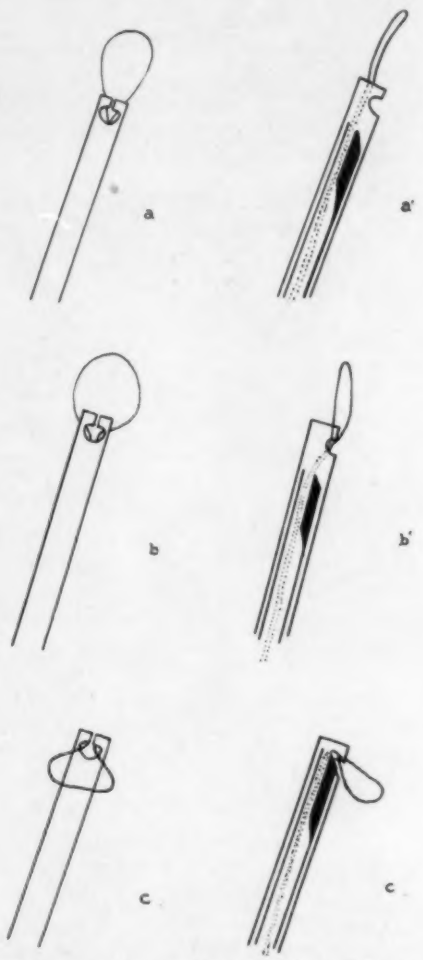


Fig. 8.—*a', b' and c'* are cross-sections at right angles to their respective numbers, *a, b* and *c*. In *a-a'*, the wire is shown passing through the lumen of the inner cannula; in *b-b'*, the wire is passed through the slit in the fenestral opening; in *c-c'*, the outer cannula is slid back and the wire loop is angulated.

over the inner cannula, *B*, with the aid of a screw, *C*, but cannot be rotated about the inner cannula. The outer cannula has a fenestral opening, preferably in the form of a heart, near its distal end. The fenestral opening communicates with the exterior by means of a slit.

Operation.—Before threading the wire loop, the two cannulas are approximated as in the position in figure 7, *II*. Then the wire loop is threaded beginning from the distal end as in figure 8, *a-a'*, through the lumen of the inner cannula and anchored to the constricting bar (fig. 7, *F*) by means of a screw, *E*. Then the size of the wire loop is adjusted to from about one-quarter to one-half inch (0.63 to 1.27 cm.) in circumference. The wire loop is then passed into the fenestral opening via the communicating slit (fig. 8, *b-b'*). The wire loop will now rest on the outside surface of the two prongs situated at each of the communicating slits.

The instrument is now ready for use. In order to angulate the wire loop, the screw, *C* (fig. 7, *I*), is turned to the right, which will move the outer cannula backward (fig. 8, *c-c'*), and when the prongs override the margin of the inner cannula, the wire loop will angulate itself.

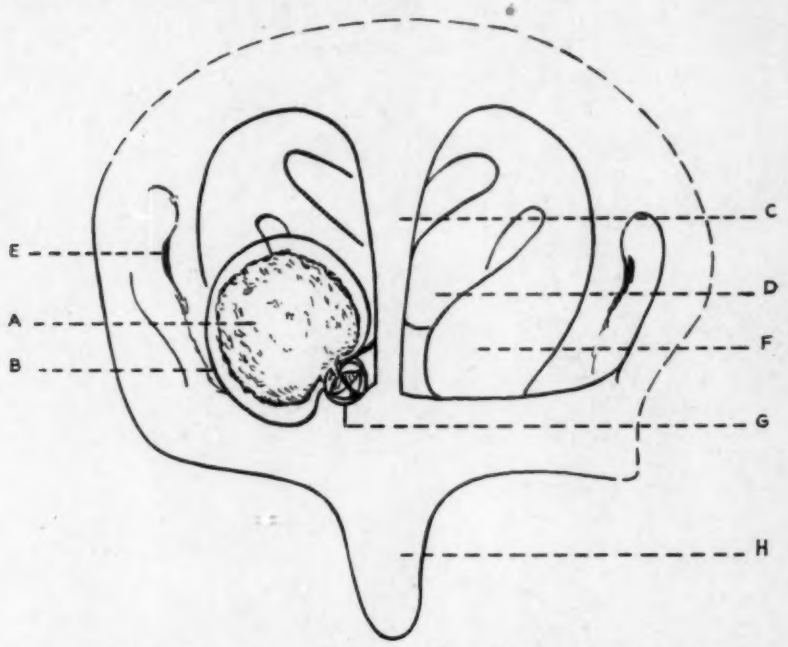


Fig. 9.—Schematic drawing of the posterior rhinoscopic picture showing the angulated wire ready to be engaged to the base of the hypertrophied tip of the inferior turbinate. *A* indicates the hypertrophied tip of the inferior turbinate; *B*, the angulated wire loop; *E*, the orifice of the eustachian tube; *C*, the septum; *D*, the middle turbinate; *F*, the inferior turbinate; *G*, the distal end of the snare; *H*, the posterior wall of the uvula.

Directions for Use.—To remove the left hypertrophied posterior tip of the inferior turbinate, pass the instrument through the left nostril alongside and parallel to the inferior turbinate, with the fenestral opening toward 3 o'clock, and to remove the right tip pass the instrument through the right nostril with the fenestral opening toward 9 o'clock until a resistance is met by the posterior wall of the nasopharynx; then slightly retract the instrument for a fraction of an inch. Turn the screw, *C* (fig. 7, *I*) to the right as far as it will go. The loop will angulate itself, and by gradually drawing out the instrument, the posterior tip of

the inferior turbinate will engage itself within the loop. Draw the instrument out until resistance is met, then hold the instrument in this position, while turning the screw, *C*, completely to the left. Then snare with the aid of the constricting bar, *F* (fig. 7, *I*). To simplify matters, a different instrument may be used for each side.

The Welch Allyn Company of Auburn, N. Y., cooperated in the mechanical construction of the lighting, catheterizing and the trocar instruments, and the Tieman Surgical Company of New York in the construction of the angulating snare. These companies are the manufacturers of the respective instruments.

736 West End Avenue.

AN UNUSUAL CASE OF TWO FOREIGN BODIES IN THE LUNG*

LOUIS DAILY, M.D., AND J. C. DICKSON, M.D., HOUSTON, TEX.

Two foreign bodies in the lung at one time are not unusual, but such an odd combination was recently found in a patient, bringing up several problems, that it was decided to report the case.

E. H., a girl, 3½ years old, in good health with an unimportant previous medical history, was brought to her physician with the complaint of a rattling, nonproductive cough of one week's duration. Examination revealed râles over the lower part of the right side of the chest, with increased breath sounds. Roentgen examination was advised, but the parents refused. One week later, the child returned. The cough was worse, and she had a daily temperature of from 100 to 101 F. Examination of the chest demonstrated harsh, bubbling râles over the lower part of the right side of the chest. The parents consented then to roentgen examination, which showed a paper clip, 2.5 cm. by 0.75 cm., on the right side, between the fifth and sixth ribs and near the edge of the sternum (fig. 1).

A bronchoscopy was performed without anesthesia or preliminary hypodermic injections. A Jackson 5 mm. bronchoscope was used. After the trachea and the bronchus were cleared of secretions by suction, the operator found a black object wedged tightly in the right bronchus at the junction of the middle and lower lobe. This was grasped with a Jackson rotating forceps and removed. When the mother saw the foreign body, she exclaimed, "There is the bead she swallowed." Three months previously, the mother thought the child had swallowed a wooden bead, but as no symptoms developed and nothing was found on examination, the incident was forgotten.

After a few minutes, the bronchoscope was reintroduced, and the paper clip was located in the right lower bronchus and removed (fig. 2). It took five minutes to introduce the bronchoscope and to remove the foreign bodies. The temperature dropped to normal, and the child left the hospital the next day. A slight complication arose on the fourth day, when acute otitis media developed on the right side. The drum was incised. The ear healed after discharging for a week. There has been no further trouble, and the child has been in excellent health.

The mother, on being questioned, remembered that the child had played with a paper clip six months before. Evidently the patient had aspirated it at that

* Submitted for publication, Oct. 4, 1930.

time. The paper clip, being small and metallic, caused no obstruction and only a slight irritation. Thus no symptoms developed. Three months later, the child aspirated the bead. It measured 1.2 cm. by 0.75 cm., and had a 3 mm. hole through the center. It evidently lodged in the lower part of the right bronchus. Again no symptoms appeared, because the air passed in and out through the hole in the

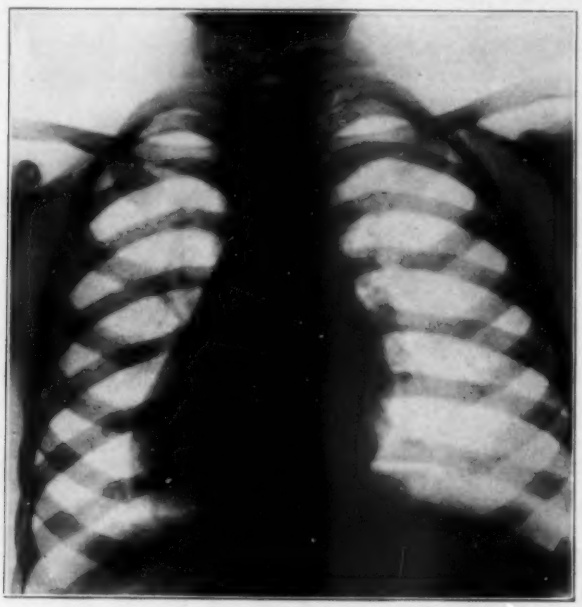


Fig. 1.—Anterior view of the chest showing the paper clip. No shadow of the bead is seen.

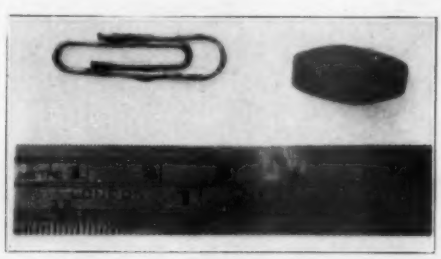


Fig. 2.—The bead and the clip removed from the lung.

center. Symptoms developed only when the bead produced enough irritation to cause bronchitis.

The case shows that one often pays too little attention to the history when the results of examination are negative. The mother was sure that the bead had either been swallowed or aspirated, but as no symptoms appeared, it was forgotten.

This also brings up the question of what might have happened if only the bead had been aspirated, or if it had gone down first, as the wooden bead does not cast a shadow on the roentgenogram. The clip might have been removed, leaving the bead in the lungs. An abscess of the lung would probably have been the result.

Undoubtedly, many cases in which a foreign body, nonopaque to the roentgen rays, is in the lung, are diagnosed as chronic bronchitis or bronchiectasis. This fact supports Jackson's claim, that in all cases of infection of the lungs that do not clear up, a bronchoscopic examination should be made, regardless of what the roentgenogram shows. When skilfully done, the procedure is not dangerous, and it certainly is of assistance in the treatment for many obscure conditions of the lungs.

Abstracts from Current Literature

Ear

LATERAL SINUS THROMBOSIS COMPLICATING MASKED MASTOIDITIS. IRWIN J. KLEIN and MAX LEDERER, Am. J. Dis. Child. **40**:1045 (Nov.) 1930.

In considering the diagnosis in obscure septic conditions, especially when an infection of the middle ear has occurred, the possibility of a lateral sinus thrombosis may be overlooked. In the case reported by Klein and Lederer, cough, fever and otitis developed in a boy, aged 7 weeks; the skin was cyanotic, the pupils were pinpoint sized and fixed. The temperature mounted to 106 F. There was one convulsion, with clonic movements of the right upper extremity and twitching of both sides of the face. At autopsy, a large hemorrhage was found on the anterior surface of the right hemisphere, and a firm thrombus was discovered in both lateral and superior sagittal sinuses. The clinical picture of this complication is not characteristic; the most important features are the high fever, the meningitis-like symptoms and the altered pressure discovered on spinal tap.

DAVIDSON, Newark, N. J.

A CASE OF HYSTERICAL MASTOIDITIS. MOUNIER-KUHN, Rev. d'oto-neuro-opht. **8**:183 (March) 1930.

A girl, aged 15, was seen on August 22. She complained of pain in the ears for the preceding ten days, fever and loss of appetite; she had lost 2 Kg. in two weeks. For the previous two days the pain had been localized in the left mastoid region. The past history elicited the following facts: Since an attack of diphtheria at the age of 10, there had been a succession of troubles. First, pain in the spine was labeled Pott's disease, and she was kept in bed for a year. This was followed by several weeks of fever and insomnia. Finally, in January, 1929, intense and continuous headaches set in. An operation on the adenoids gave relief for a time, but the headaches returned and visual difficulties appeared. Glasses brought renewed relief until the beginning of August, when there was a complaint of pain in the ears and fever. One brother was afflicted with pulmonary tuberculosis.

Examination revealed a normal right ear; the left tympanic membrane was barely rosy, and the left mastoid was sensitive at the tip. The spinal column was supple, and there were no signs of meningitis or vasomotor troubles. The patient was poorly developed. Paracentesis of the left drum was not followed by a discharge, but relieved the pains temporarily. The pain returned, accompanied by fever and complaint of poor vision in the left eye. The spinal fluid was normal. An operation on the mastoid revealed no pathologic involvement, but relieved all complaints for three weeks. Then there were complaints of pain in the right ear, fever and insomnia. A neurologist was consulted and advised a return to the family life and occupation. Again there was amelioration for a while, and again the pain in the right ear and fever returned. An injection of procaine hydrochloride over the mastoid gave relief for a week, and then distilled water was equally efficacious. Finally, a pediatrician was called in who was firm enough to impress the patient, and all complaints ceased.

The basis of this complex pathologic process was evidently the emotional shock from the diphtheria. Contrary to previous ideas, the psychic shock of the operation was not sufficient to effect a cure, and recourse to energetic psychotherapy was necessary.

DENNIS, Colorado Springs, Colo.

AURICULAR TETANUS AND ITS RELATION TO OCULAR TETANUS. G. V. T. BORRIES, Rev. d'oto-neuro-opht. **8**:423 (June) 1930.

Auricular tetanus is not a particular disease, but the term applies to cases arising from the ear. While it is rare, it probably is more frequent than is

supposed. Permin reported 371 cases of tetanus in Denmark, among which there was 1 case of auricular tetanus and none of ocular tetanus. The author was able to collect only 12 cases. One case occurred in a boy who was admitted to the hospital with the diagnosis of otitis with cerebral complication. Eight days before admission, a piece of wood had penetrated the left ear. The symptoms were pains in the ear and the back, trismus, convulsions and opisthotonos. The splinter was found in the ear, where it had caused injury of the anterior wall of the canal. Death occurred on the following day.

The symptoms of tetanus are not unlike cerebral complications of otitis; the stiffness of the neck suggests meningitis; torticollis is seen in tetanus and also in abscess of the neck of otogenous origin; Oppenheim has shown that trismus is found also in tuberculous meningitis affecting the two central gyri. The facial paresis characteristic of tetanus may also lead to an error in diagnosis. Other clinical peculiarities may make the diagnosis difficult; either an otogenous cerebral disease may exist with a picture of tetanus or an aural suppuration itself may cause tetanus. Moulonguet and Perrier observed a case of lateral sinus thrombophlebitis with trismus, contraction of the muscles of the neck and a positive Kernig sign in which tetanus was diagnosed and serum was given. After removing the thrombus, however, the patient recovered.

Auricular tetanus can appear without traumatism in a suppurating ear. Knud and Schroeder reported a case of prolonged otitis in which the tetanus was induced by scratching the canal with a hairpin. Meseck and Klestadt each reported a case of suppurating otitis complicated by tetanus in which a tampon of cotton soaked in pus, containing tetanus bacilli, was removed from the auditory canal. It is probably true that such an infection can arise in the cavity after a radical operation has been performed on the mastoid, although no case has as yet been observed.

Auricular tetanus occurs with the picture of cephalic tetanus, with trismus, dysphagia and at times a disease of the facial nerve, either a spasm or a paralysis. This latter may, however, be lacking and is certainly rarer in auricular than in ocular tetanus, a fact which has some diagnostic importance. There seems to be no difference in principle between the manner in which the facial nerve and the other nerves are attacked in tetanus, but the facial nerve is perhaps more susceptible and its symptoms are more striking. In partial tetanus, the disease of the facial nerve may be either a paralysis or a spasm. In complete tetanus, the participation of the facial nerve is almost always in the form of a spasm. Other nerves may also be paralyzed in partial tetanus: the oculomotor, the trochlear, the hypoglossal and, perhaps, also the trigeminus. These local paralyses are easily overlooked because they are hidden by a spasm of the contiguous muscles. The same toxin can produce a spasm as well as a paralysis, and a single nerve can be particularly vulnerable.

A case is described in which the patient with facial paresis accompanying a suppurating ear was sent from the ophthalmologic to the neurologic clinic and from there to the otologic clinic. In the last, exenteration of the mastoid was done, and only later was it discovered that the case was tetanus from a previous lesion of the eye which had not been known.

In diseases of the facial nerve without apparent cause, tetanus must be thought of even if there is no history of traumatism. Likewise, when, after a traumatism of the ear or of the eye, a paralysis appears, it must be remembered that the latter is not always a consequence of trauma, but that it may also be a sign of tetanus.

DENNIS, Colorado Springs, Colo.

THE TREATMENT OF VERTIGO AND TINNITUS BY THE ELECTRO-ANESTHETIC CURRENTS OF ARAYA. G. WORMS, Rev. d'oto-neuro-opht. **8**:442 (June) 1930.

This report embraces twelve cases with varied aural pathologic conditions: seven cases of otosclerosis and cicatricial otitis; two cases of otospongiosum; one case of trauma (explosion); one case of chronic suppurating otitis media and one case of tinnitus without auricular lesion. In the last five cases the treatment was

without effect. In the first seven the results were: marked relief from vertigo; complete disappearance of tinnitus in three cases; improvement in three cases, and no effect in one. The deafness was improved in three cases. In all cases there was no active lesion of the labyrinth.

DENNIS, Colorado Springs, Colo.

LYMPH CHANNELS CONNECTING THE MIDDLE EAR WITH THE CRANIAL CAVITY. D. TANTURRI, Monatschr. f. Ohrenh. **64**:1005 (Sept.) 1930.

Tanturri points out that the anatomic study of clinical cases observed during the last twenty years confirmed his statements made in 1911 with regard to the paths of communication of the middle ear with the cranial contents. The experiments aimed to ascertain the way of transmission of an infection from the middle ear to the meninges. The researches indicated that besides a direct communication of the middle ear with the cranial contents, there is an indirect one by way of the labyrinth. The direct communication was demonstrated by the fact that the dye injected into the perineurium of the facial nerve appeared in the perifascicular tissue. Evidently, the facial nerve presents a path connecting the middle ear with the cranial contents. An infection is apt to spread along the nerve in the internal auditory meatus, up to the contents of the posterior cranial fossa, by way of the perineurium or the lymph passages of the nerve. Accumulation of pus in the facial canal, caused by a pathologic process in the wall, brings about compression of the perineurium and paralysis of the nerve. The necessity for surgical intervention, consisting in elimination of the pressure, is urgent in such cases; it results in recovery. Transmission of an infection from the middle ear to the cranial contents by the way of the facial canal is rare; it occurs through the lymphatic spaces of the nerve. The indirect communication of the middle ear with the cranial contents was demonstrated experimentally. An injection of india ink through the membrane of the fenestra rotunda readily penetrated into the interdural spaces. The dye was absent in the surrounding spaces of the semicircular canals and of the utricle; it was present in the perilymphatic spaces of the cochlea, up to the region of the saccule. Also the cochlear canal, the spiral canal of the modiolus and the canaliculi of the lamina spiralis remained free from the dye. The results showed that the dye had spread by way of the perilymphatic spaces, whereas the endolymphatic spaces proved not to be involved. Shortly afterward Kasai found that an injection of streptococci into the subarachnoid space was followed by infection of the labyrinth. This developed by way of the perilymphatic spaces; the endolymphatic infection occurred later. While hydrops is induced merely by irritation of the lymphatic and perilymphatic system, serous labyrinthitis is caused by the transmission of bacilli and toxins through the lymph passages of the mucosa. The perilymphatic spaces and the aqueduct of the cochlea are the route of transmission from the labyrinth to the cranial contents. Direct communication of the subarachnoid space with the perilymphatic spaces, through the aqueduct of the cochlea, was verified by many authors. Shambaugh found vascular anastomoses between the middle ear and the labyrinth in calves. Mouret's statement that there are no lymph vessels in the temporal bone, running along the haversian canals, was confirmed by Tanturri. The injected dye penetrated into the vascular wall, but did not enter the mucosa or the bone of the middle ear. Another series of researches demonstrated that the arachnoid, involved in the sheath of the auditory nerve, does not extend to the base of the internal auditory meatus. Injection of a dye into the cochlear nerve, by the subarachnoid route, resulted in staining of the modiolus canaliculi and of the spiral canal of the modiolus. This route of transmission of an infection from the labyrinth to the cranial contents is important; serofibrinous, and especially suppurative, inflammations of the labyrinth spread to the internal auditory meatus and to the meninges by the way of the auditory nerve. The conclusions were drawn that the route of communication between the middle ear and the cranial contents is not represented by the lymph vessels, but by the lymphatic spaces and the lymphatic interspaces, which include the lymph passages of the facial

and auditory nerves and the perilymphatic spaces of the cochlea and, more rarely, of the vestibule. Thus, besides vascular connections between the tegmen tympani and the sigmoid sinus, canalicular formations and ways created by pathologic processes, the spread of an inflammation from the middle ear and the labyrinth occurs also by the way of perilymphatic spaces and nerve sheaths. The perilymphatic system of the internal ear and the lymph spaces of the facial and auditory nerves are the passages for the spread of an infection in the middle ear; they form a part of the lymphatic system in the human organism.

EDITOR'S ABSTRACT.

DISTURBANCES IN EQUILIBRIUM RELATED TO BILATERAL DESTRUCTION OF THE VESTIBULAR APPARATUS. J. HERZFELD, Monatschr. f. Ohrenh. **64**:1035 (Sept.) 1930.

The case described was first observed by Herzfeld in 1900. The patient, a boy, who was then 9½ years of age, had contracted scarlet fever. Pain developed in both ears three days after the beginning of the disease; soon a purulent discharge began, bringing about complete deafness a day later. A rigid, masklike aspect of the face appeared about eight days afterward. Consciousness was never disturbed; there was no involuntary micturition of defecation. From the patient's history, it appeared that there was a transient dizziness. Involvement of the two labyrinths occurred almost simultaneously. The author examined the patient six weeks later. Both ears proved to be filled with a fetid discharge; total deafness and bilateral paralysis of the facial nerve were present. When the patient kept his eyes open, he walked normally. When a radical operation was performed, a greater part of both mastoid processes was found to be reduced to sequestrums; erosions were found in the horizontal semicircular canals. The two facial canals were necrosed, especially the left. On the thirteenth day after the operation a sequestrum of two and a half semicircular canals was removed from the left ear. The healing of the surgical wounds was complete in a few months. Under the effect of extensive electrotherapy, the functioning of some of the branches of the facial nerve became restored, and the facial expression improved. The patient was reexamined eight years later. When his eyes were closed, phenomena of disturbed equilibrium were manifest, which persisted up to the time the article was written. The patient was examined repeatedly during the next twenty years. The bone conduction and the air conduction of both cochleae were completely excluded. Lateral movements of the eyes induced a horizontal or a slightly rotatory nystagmus which did not change on rotation of the head; it was not of labyrinthine origin. Compression or galvanic currents were without influence on the nystagmus. This observation showed that the cochlear and the vestibular portions of both labyrinths were destroyed. During tests made when the patient's eyes were open, his static and dynamic behavior appeared normal. The results of examination of the static behavior of the patient when his eyes were closed varied. When he was examined in 1909 and 1913, swaying had not developed when he jumped forward and backward; the gait was not straddling, and he had no difficulty in standing on one leg. When he was examined in 1908, 1917, 1928 and 1929, with his eyes closed, there was a straddling gait; standing on one leg was difficult and was accompanied by a considerable swaying. The disturbances in equilibrium appeared to depend also on the general condition; they became more pronounced, for instance, in a state of fatigue or of irritation. The swaying was marked during tests with a goniometer, especially with a spring mattress. Neurologic exploration indicated that the cerebellum, the pons varolii and the medulla oblongata were not involved. The physiologic reflexes were normal; there were no pathologic reflexes. When the patient's eyes were closed, the disturbances in equilibrium were observed only in an upright position; they were absent when the patient was sitting or lying. Adiadokocinesis, past pointing and disturbances of sensibility or of the appreciation of weight were absent. The grasping force of both hands was equal. The author was convinced that the disturbances of

equilibrium were present at the time of the first examination; they were not revealed then because the examination was incomplete. He disagrees with the investigators, who think that a bilateral destruction of the vestibular apparatus does not necessarily result in a disturbance of the equilibrium.

EDITOR'S ABSTRACT.

USE OF SPACE ANALYZER FOR EXAMINATION OF DEAF-MUTES. A. G. FETISSOW, Ztschr. f. Hals-, Nasen- u. Ohrenh. **26**:351 (Aug. 9) 1930.

The apparatus consists of a cubiform chamber, 1.25 meters high. By means of cords, it is attached to an iron ring, provided with a loop, which is held by a hook fixed in the ceiling. Rotation of the iron ring results in twisting of the loop. A bench on which the patient sits is placed in the middle of the chamber; the latter is kept on a stand supplied with a lever. The chamber is raised by pushing down the lever; by pressing on the pedal, the stand is brought back to the floor, and the chamber remains suspended in the air. The chamber starts to turn around when the loop is twisted. The details of the apparatus and the procedure of the examination are given. Vestibular functioning was previously estimated by the caloric and the rotation tests. The development of the sensation of rotation is connected with an isolated irritation of the semicircular canals. First fifteen persons with normal hearing and an intact vestibular apparatus were examined; the sensation of rotation appeared in them from 1.5 to 3 seconds after the beginning of rotation. The average of the latent period was 2.24 seconds, while the speed of rotation of the chamber amounted to four circles in 40 seconds. Hence, the sensation of rotation developed at the beginning of the first circle when the speed of rotation was sufficient to cause an irritation of the semicircular canals. At the beginning of the fourth circle, when the speed was notably decreased, the sensation of rotation reappeared, but this time rotation appeared to be in the opposite direction. During the rest of the time, the patient had the impression that the chamber stood still. Evidently not the rotation itself but its speed is an important factor in irritation of the semicircular canals. Forty-nine deaf-mutes were next examined; in thirty-five the vestibular irritability was absent on both sides. They had no sensation of rotation while they were in the chamber, but believed that it was standing still. A few of them had the sensation of a slight swinging motion at the time when the chamber stopped. In fourteen deaf-mutes with positive caloric and rotation reactions, the latent period appeared prolonged to from 9 to 13 seconds. The average of the latent period was 10.85 seconds. The sensation of rotation developed at the end of the first circle, when the rotation speed of the chamber was considerable. Obviously a much stronger irritation of the semicircular canals is required in deaf-mutes than in normal persons. Fetissow stresses that the number examined was limited, because no children under a certain age can be examined in the chamber.

EDITOR'S ABSTRACT.

Pharynx

TUMORS OF THE RHINOPHARYNX WITH CRANIAL PROPAGATION: THEIR EARLY DIAGNOSIS AND THE IMPORTANCE OF ROENTGENOLOGIC EXAMINATION. G. WORMS and R. CARILLON, Rev. d'oto-neuro-opht. **8**:161 (March) 1930.

Six well studied and fully reported cases prompted this article. When tumors of the rhinopharynx begin in the peritubal region they rarely show characteristic signs of their presence. The patient usually exhibits an ordinary pharyngitis until some complication arises, which is almost always a disease of a nerve. These tumors grow by preference toward the skull along the course of the eustachian tube and enter the cranial cavity by erosion of the bone rather than by infiltration or by way of the basal foramina. Paralysis of the trigeminus is an early sign, beginning with pain, which is of moderate severity and is continuous. The superior and inferior maxillary divisions are affected before the ophthalmic, and are tender

to pressure over their points of emergence. The motor fibers participate in the disturbance, indicated by atrophy of the masticator muscles and by an oval oblique shape of the mouth, caused by projection of the inferior maxilla toward the healthy side.

Great stress is placed on the value in diagnosis of careful roentgenologic studies of the cranial base. According to Hirtz' technic, the tube must be centered exactly over the petrosphenoidal region of the suspected side. Loss of detail of the structure of the point of the petrous pyramid, veiling of foramina in the middle fossa and abnormal transparency of the bony mass eroded by the neoplasm may be seen. At a later stage, a distinct lacuna, usually limited behind by the superior crest of the pyramid, will be found. Sometimes the shadow of the tumor in the nasopharynx can also be made out.

In the early stages, enlarged glands in the retromaxillary or submaxillary angle may be felt, and at times they are even found on both sides.

Subcranial neoplasms, which arise from the mucosa or the adjacent lymphoid structures, are epitheliomas or lymphosarcomas, while those arising in the bone or degenerated dura are osteosarcomas, fibrosarcomas, psammonas, meningiomas, etc.

To the trigeminal paralysis is soon added paralysis of the abducens nerve, and the syndrome of the petrosphenoidal junction is complete. Later on, the syndrome of the cavernous sinus develops. More rarely the posterior fossa is invaded and facial, vestibular and pontile symptoms appear. In one of the cases the facial nerve was paralyzed from erosion and extension of the tumor into the tube, the middle ear and its adnexa. The anterior fossa is still more rarely invaded. The anosmia complained of is caused by mechanical obstruction of the nares.

Extension of the growth laterally results in bilateral paralysis. In one case both fifth and sixth nerves were involved. The spinal nerves are almost always spared, although in one case paraplegic phenomena were explained by a vertebral metastasis. Exceptions to the rule that these tumors do not cause increase of intracranial pressure were found in two cases, for both showed papillary stasis and one required decompression. Involvement of the sinuses, orbit and nasal fossae is a terminal phenomenon. Death occurs in from six to twenty-four months.

DENNIS, Colorado Springs, Colo.

TONSILLECTOMY DURING OR SHORTLY AFTER AN ACUTE INFECTION OF THE TONSILS. S. LEVINGER, München. med. Wchnschr. **77**:1666 (Sept. 26) 1930.

Acute infection of the tonsils has generally been considered a contraindication for tonsillectomy. The danger of sepsis, pyemia and deep-seated phlegmonous infiltration of the neck are complications which have frequently been reiterated. Levinger does not agree with this attitude. He has performed tonsillectomy under local anesthesia in 235 cases of peritonsillar abscess during the acute stage. In 60 cases the abscess was preceded by typical angina. A few days after the fever and general symptoms had subsided, the author removed the tonsils. There were no complications. Early operation prevents the recurrence of the abscess and obviates a second operation (tonsillectomy), which is necessary if the abscess is incised and drained according to the present custom. It is important to follow the author's technic in detail. He believes that the removal of the lower pole of the tonsil is the cause of the many complications that have been described.

BRAHDY, Mount Vernon, N. Y.

Nose

RESULTS OF ENDONASAL OPERATIONS ON THE ACCESSORY SINUSES. W. KRAINZ, Monatschr. f. Ohrenh. **64**:907 (Aug.) 1930.

Hajek's endonasal method consists in removing diseased posterior cells of the ethmoid sinus and in rendering the nasofrontal duct accessible; with this method, the exposure of the sphenoid sinus is easy. On the other hand, the disadvantages

of the method consist in the fact that the anterior cells of the ethmoid sinus cannot be reached and therefore recurrences are frequent. Partial resection of the middle turbinate bone is sometimes followed by atrophy of the mucosa; besides, the frontal sinus is inaccessible for inspection. Halle's method, by removal of the agger nasi, enables one to inspect the ethmoidal labyrinth up to the base of the skull. Removal of the middle basilar portion with a chisel renders inspection of the frontal sinus possible; then insertion of a mucosal flap in the restored nasofrontal duct ensures permanent opening of the sinus and enhances the results of the operation. Halle's method makes possible conservation of the middle turbinate bone; it protects the lamina cribrosa against injury during operation and prevents atrophy of the nasal mucosa after operation. The results obtained with Caldwell-Luc's and Denker's methods were identical with those obtained with the endonasal method of Sturmann and Canfield. Normal alimentation after operation is the advantage of the latter method, while an incomplete elimination of the mucosa is its disadvantage.

The results reported by Krainz refer to 162 patients, 90 men and 72 women, who were treated from 1925 to 1928 in the University clinic of Innsbruck. While Sturmann and Canfield's method was used for operations on the antrum of Highmore, Halle's method was used for operations on the upper sinuses. The diagnosis of a chronic disease of the frontal or of the ethmoid sinuses was based on the history of a persisting headache, a purulent discharge and disturbance of nasal respiration. The finding of polypi and pus in the middle nasal meatus was evidence of the correctness of the diagnosis, which was confirmed by Killian's exploration of the middle nasal meatus and of the rima. The presence of polypi in the ostium maxillare pointed to disease of the antrum of Highmore; the disease was considered as chronic when the suppuration did not subside in response to irrigations. The irrigations were made with Siebenmann's cannula, which was introduced in the middle nasal meatus; the irrigations never caused any complication. A 3 per cent solution of cocaine was used in anesthesia for exploration of the ethmoid bone or for irrigation of the antrum of Highmore. In cases of disease of the antrum of Highmore, the results of roentgen examination were confirmed at operation in about 75 per cent; in cases of disease of the frontal sinus, the results agreed in scarcely 60 per cent, and in cases of disease of the sphenoid sinus agreement was found in less than 10 per cent. Hence, the value of roentgen examination consisted chiefly in demonstration of the sinuses, especially the frontal sinuses. The treatment was conservative when the purulent discharge from the upper sinuses was not associated with the presence of polypi on the mucosa. It consisted in anematization of the mucosa in the middle nasal meatus, in stretching the middle turbinate bone and in application of light treatment to the head. In patients more than 50 years of age, an operation on the upper sinuses was rarely performed (only in 18); when intervention was undertaken it was usually limited to relieving the obstruction to nasal respiration, such as polypi. Most of the patients operated on were from 30 to 50 years of age; only 21 patients were under 20. A unilateral pansinusitis was observed in 66 per cent, while a bilateral pansinusitis was observed in 23 cases. A bilateral disease of the frontal sinuses occurred in 20 per cent and a bilateral disease of the antrum of Highmore in 16 per cent. An isolated suppuration of the antrum of Highmore was noted in 23 per cent; in 11 per cent, there was a disease of the frontal sinuses and of the ethmoid bone alone. All operations on the ethmoid and the frontal sinuses were preceded by a submucosal resection of the septum, within from eight to ten days. The endonasal operation was always performed under local anesthesia. (The technic used for anesthesia as well as that for the operation is given.) The anesthesia proved to be sufficient to allow the use of a chisel and a file for the bones. In 114 cases, the upper sinuses and the antrum of Highmore were operated on simultaneously; in 165 cases, the endonasal route was used for operation on the antrum. The duration of an operation on the antrum never exceeded half an hour; it lasted from forty-five minutes to an hour in cases of pansinusitis. The postoperative treatment consisted in the oral administration of 4 Gm. of methenamine on the day of the operation, besides the application of a four-tailed

bandage to the nose and of one or two icebags. Methenamine is given for the purpose of preventing a meningeal irritation from manipulation of the top of the ethmoid bone. Thus, mild meningeal phenomena occurred only in 2 cases; they developed on the second day after operation and disappeared as soon as the tampon was changed in the ethmoid bone. Postoperative pains were insignificant; the use of amidopyrine or of a barbital derivative was required the night of operation in exceptional cases. There were sometimes local reactions, as swelling of the upper eyelid and of the superciliary region, which on the application of a new dressing or moist and warm poultices usually subsided within from four to six days. In 3 cases, the swelling of the cheek was pronounced; it ended in an abscess in one instance. In cases of operation on the antrum of Highmore, the swelling of the cheek was notably less when the endonasal and not the oral method was used. The tampons were left for two days and were removed without causing pain, especially if they were coated with a boracic ointment. (The details of the postoperative treatment are described.) In 10 per cent of the cases (most of them in women) in which the secretion persisted and the healing was deficient, protein therapy was successfully used in the form of from 8 to 10 intramuscular injections of a casein solution. The injections, each containing 1 cc., were given at two day intervals. In 46 cases in which operation was performed on the antrum of Highmore, the test of faradic irritability indicated that the dental nerves remained intact in 16; in the remainder, from 1 to 4 dental nerves appeared injured. The injury proved to be transient. The results of the test were about the same whether the endonasal or the oral method was used. A primary healing was observed in 75 per cent of 148 patients operated on for disease of the frontal sinuses and the ethmoid bone. In 12.5 per cent, the patients left the hospital with a persistent discharge from the frontal sinuses. In 8.5 per cent, a second operation by the endonasal route was required on account of polypi which developed a few weeks after the first intervention. In 4 per cent, a radical operation was necessary after previous endonasal intervention. A primary healing was observed in all but 1 of 165 patients who were operated on for disease of the antrum of Highmore. About 48.5 per cent of the patients operated on endonasally for disease of the frontal sinuses and the ethmoid bone were reexamined from one to three years after the operation. Morbid subjective signs had vanished in all; in 72 per cent, the healing persisted. In 28 per cent, polypi developed around the opening of the frontal sinuses; there was also an edematous thickening of the ethmoid mucosa and a slight mucopurulent discharge. About 54 per cent of the patients operated on endonasally for disease of the antrum of Highmore were reexamined; the healing appeared to be permanent in all.

Krainz concludes that Sturmann's method is preferable to Luc's and to Denker's methods, because the former is more convenient for the patient, while the results are analogous. Further, the results obtained with Halle's method are compared with those obtained with Hajek's method. Of 174 patients operated on for disease of the frontal sinuses and of the ethmoid bone by Hajek's method, a second operation was required in 21 per cent (8.5 per cent with Halle's method) and a radical operation was required in 8 per cent (4 per cent with Halle's method). The author agrees that Halle's and Sturmann's technics are more complicated than those of other operations; that is, they require extensive experience. In unfavorable cases, the exposure of the dura at the top of the ethmoid, outward from the lamina cribrosa, may be followed by an endocranial complication. In rare cases, anastomoses of the olfactory fibers may be present in the duct of the frontal sinuses; injury of these fibers and of the lymph sheaths is a direct menace to the meninges. Krainz emphasizes that such an anomaly is exceptional.

EDITOR'S ABSTRACT.

RESULTS OF SURGICAL CONSTRICTION INDUCED FOR TREATMENT OF OZENA. A. BLAU, Monatschr. f. Ohrenh. **64**:929 (Aug.) 1930.

Postinflammatory atrophy of the turbinate bones and of the mucosa is the chief cause of the morbid phenomena observed in cases of ozena. Improvement of the atrophy and restoration of a normal nasal cavity and a normal nasal secre-

tion, owing to an increased supply of blood to the turbinate bones, are the purpose of operation. An injury of the nasal mucosa results in the formation of crusts on the scar. Such crusts proved to be especially extensive after removal of the middle and the inferior turbinate bones; hence conservation of an intact nasal mucosa is necessary for the success of an operation. Of Blau's patients with bilateral ozena, 150 were operated on by the combined Lautenschläger and Hinsberg's method (displacement of the lateral nasal wall and the use of absorbable magnesium plate sutures). The plates were left in the antrum of Highmore; the pressure of the plates never caused necrosis. Suppuration of the antrum developed in a few cases; it subsided after the institution of irrigation. After the operation, there was always considerable swelling of the cheeks and fever, which persisted for a few days. Meningeal signs, abscess of the cheek or suppuration of the lacrimal sac never developed. The operation was performed under local anesthesia, being preceded by the administration of morphine and scopolamine, except in 2 children. One hundred and twelve patients were reexamined later. In 6 the operation had to be repeated; the results were good in 1. In the remainder, failure was due to injury of the mucosa at the lateral wall. Recovery was observed in 42.8 per cent; improvement in 39.8 per cent, and failure in 18 per cent. Transplantation of fat tissue and fascia was carried out in 13 patients. The operation was successful in 2; therefore, implantation through an incision of the septal mucosa was performed in 1, and through the oral route (Bureck) in the other. Eckert and Mobius's method (implantation of fine pieces of bone of the restored bone, beneath the septal mucosa on one side) was used in 9 patients. The operation failed in all cases, since the implant was expelled after weeks, or even months. Therefore, the method was discontinued. Blau points out that surgical treatment is symptomatic and not causal.

EDITOR'S ABSTRACT.

INSTRUMENT FOR COSMETIC OPERATIONS ON the NOSE. W. WOJATSCHEK, Monatschr. f. Ohrenh. **64**:1066 (Sept.) 1930.

For cosmetic operations on the nose, Wojatschek suggests a straight saw that somewhat resembles Joseph's saw. With such a saw, the force of the manipulations is not decreased by the principle of the lever. It can be used for subcutaneous operations on the lateral wall of the nose, because the lower half of the nose (the tip of the nasal pyramid) can easily be shifted, during sawing, to the side of the operation. The sawing line and the wound of the nostril can thus be retained at a direct line with the longitudinal axis of the saw. The straight saw can be used for resection of a nasal hump; resection of the cartilaginous portion of the hump, usually made with the knifelike device with which Joseph's saw is provided, is easily removed with the straight saw, especially when the incisions for orientation were made with a scalpel. The cartilaginous portion can be removed with a scalpel or with scissors. The straight saw can also be used for detachment of the tibial edge, for sawing the medial wall of the antrum of Highmore during an endonasal operation and for removal of an ossified thyroid cartilage in cases of laryngeal fissure. The author stresses the fact that severing with a saw gives a better appearing result than is obtained with a chisel, and therefore is more appropriate for cosmetic purposes. The straight saw is further recommended for removal of crests and of excessive bony parts developed at the nasal base.

EDITOR'S ABSTRACT.

TWO CASES OF NASAL GLIOMA. H. ADLER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **26**:305 (Aug. 9) 1930.

One of Adler's cases occurred in a girl, aged 19. Impairment of the nasal respiration, especially on the left side, started a year prior to admission to the hospital. It was accompanied by a swelling under the left eye, frequent nosebleed and a feeling of dryness in the mouth and in the throat. The disturbances were not relieved by repeated surgical interventions. On examination, besides a

left suborbital swelling and tenderness of the orbital margin, deviation of the nasal septum to the right resulting in obstruction of the right middle nasal duct was found. The left nasal meatuses appeared to be filled with a red tumor. On roentgen examination, shadows corresponding to the ethmoid bone, antrum of Highmore and frontal sinus were found on the left side. Luc-Caldwell's operation was performed under local anesthesia. Most of the left antrum of Highmore was filled with a soft tumor which apparently originated in the ethmoid bone. The antral mucosa proved to be intact. The tumor included cheesy particles and a large number of fragments of bone. Healing occurred without any complications. On microscopic investigation of the tumor, it was found that there was blood beneath the ciliated epithelium; the submucous connective tissue contained a solid mass in the form of cords and a soft mass in the form of nests. Under low magnification the solid mass seemed to be sarcoma, while under high magnification the cells of the cords and of the nests proved to consist of round nuclei, occasionally surrounded by a protoplasmic areola. The nuclear formations pointed to neuroglia; they resembled those described by Schaffer in cases of adendroglioma or oligodendroglioma. The presence of intercellular fibrils forming a fine reticulum increased the resemblance to a glioma. The presence of blue homogenous masses indicating calcareous degeneration was another characteristic of the tumor. The calcium formed a border around the cellular nuclei or was agglomerated in clumps. The second case was observed in a woman, aged 24. The patient's history included an operation performed eight years before on account of disturbed nasal respiration. The intervention brought about relief lasting for seven years. Within the last year, nasal respiration, especially through the right half of the nose, became more difficult; nosebleed appeared frequently. On examination a slight swelling was found under the right eye; the right orbit was somewhat tender. The nasal septum proved to be deviated, thus obstructing the lower nasal duct on the left side. The right inferior meatus of the nose was filled with a tumor connected with the turbinate bone. At operation, the anterior cellular part of the ethmoid bone proved to be filled with the tumor, while the posterior cellular part was filled with pus. The mucosa remained intact therein. No masses of tumor were found in the antrum of Highmore. (The details of the operation and of the postoperative treatment are given.) Nasal respiration became completely restored after the operation. The microscopic features of the tumor in the second case remarkably resembled those in the first case. In both cases, the condition is considered a nasal glioma with considerable regressive changes in the form of calcification. There was no recurrence after five years in one case and after six months in the other. Metastases were absent. The author cites many theories as to the pathogenesis of nasal glioma. It is presumed that local dystrophic disturbances, as well as calcification associated with degeneration of tissues, might have played an important rôle in the considerable deposit of calcium noted in both cases.

EDITOR'S ABSTRACT.

Miscellaneous

NINE CASES OF OTOGENOUS ABSCESS OF THE BRAIN. A. REJTÖ, Monatschr. f. Ohrenh. **64**:1174 (Oct.) 1930.

In nine patients with otogenous abscess observed by Rejtö, the temporal lobe was involved; sometimes the abscess extended to the occipital lobe. Eight of the patients were males and one a female; their ages ranged from 7 to 42. In three instances, surgical intervention took place as soon as signs of cerebral abscess developed; the patients recovered. The other six patients were admitted to the hospital with a pronounced meningitis; all of them died. Four patients were observed for from sixteen to twenty-eight days before signs of a cerebral abscess became manifest. In one case general tuberculous symptoms prevailed at first; at the fourth week an ataxic gait and incoordinated movements of the arm appeared pointing to a cerebral abscess. In another case, mental disturbances were the

predominant feature in the beginning; later sudden appearance of a severe headache and slowing up of the pulse suggested the diagnosis of a cerebral abscess. In a third patient, severe headache, slowing up of the pulse and sudden diplopia ascertained the diagnosis of cerebral abscess; in the fourth case, a slow pulse, ataxic speech, spontaneous nystagmus and incoordination of movements of the hand confirmed the diagnosis of a cerebral abscess. All patients admitted in the manifest stage presented meningeal signs. In these cases, the dura mater of the middle cranial fossa was exposed; puncture of the dura confirmed the presence of an abscess. In the nine cases, the suppuration was chronic; there was an acute exacerbation of the chronic otitis in seven. The acute exacerbation was characterized by headache, pain in the ear, also frequently by fever; congestion of the tympanic membrane and of the tympanic cavity, sometimes tenderness or swelling of the mastoid process were found. The acute exacerbation was associated with acute nephritis in one instance and with endocarditis in another. Cerebral abscess may develop from a contact infection, whereby lesions of the bone lead to meningitis, or from encephalitis caused by a retrograde septic thrombosis in the veins of the middle ear. In the three patients who recovered, the abscess developed by the venous route; it was located in the white substance. In one of these cases Lemaitre's operation and drainage were used; in another the abscess was incised and iodoform gauze was inserted. The tampons were first changed daily, for eight successive days; then four times, at intervals of a day. In the third case, the tampons were renewed only twice. There was evidence that the results depended more on the patient's condition at the time of the operation and on the strength of his resistance than on the surgical method used.

EDITOR'S ABSTRACT.

OBSERVATIONS ON VESTIBULAR NYSTAGMUS IN MIXED NITROUS OXIDE AND OXYGEN ANESTHESIA. G. KELEMEN and D. KLIMKÓ, Monatschr. f. Ohrenh. **64**:1197 (Oct.) 1930.

Kelemen and Klimkó's observations were made on patients exposed to an operation in the abdominal cavity or on the limbs. All thirty-six patients presented an intact auditory apparatus. Among them were seventeen men and nineteen women; their ages varied from 12 to 63. The anesthesia lasted from five seconds to an hour. There was proof that no correlation exists between the duration of the anesthesia and the intensity of the ocular phenomena. During the anesthesia, the eyeballs rested in central position in 75 per cent of the cases; in the remainder, pendulum-like movements of the eyeballs developed. The movements were lateral or were directed from up and outward to down and inward. The movements of both eyeballs were associated. In one instance, nystagmus appeared before the operation, it disappeared during the operation and reappeared ten minutes after awakening. After the anesthesia, nystagmus appeared spontaneously in twenty-six patients; the direction, the intensity and the duration of the nystagmus revealed dissociation of the eyeballs. The nystagmus differed when the patient was in the recumbent or in the sitting position; it also varied with the attitude of the head. In the majority of the cases, the nystagmus appeared as soon as inhalation of the gas was discontinued; in the others, it appeared from six to thirty minutes later. The nystagmus persisted for from a few hours to over three days; usually it ceased earlier on one side than on the other side. In one case the nystagmus on the left side vanished after five and one-half hours and that on the right side after fourteen and one-half hours. During the anesthesia, the reaction to irrigation of the ears with 200 cc. of water (from 25 to 27 C.) always failed; it was always present after the anesthesia. The appearance of pendulum-like movements during anesthesia, when the loss of sensibility was complete, testified to an irritation of the labyrinth. This irritation was evidently different from the caloric irritation, because the latter was then absent. According to the theory that the functioning of the labyrinth persists during anesthesia, it is considered that the nystagmus which in one case disappeared during anesthesia and reappeared afterward was of supralabyrinthine origin.

METHOD OF SURGICAL INTERVENTION ON THE ANTRUM OF HIGHMORE. K. PETER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **26**:340 (Aug. 9) 1930.

The incision usually made for exposure of the antrum of Highmore consists in severing the buccal mucosa from the fold of the second molar to the lateral incisor or even to over the median line. With preferred method of primary sutures, the healing of the surgical oral wound by first intention often fails, because rupture of the suture is a frequent complication. Thence epithelization of the margins of the wound results in the formation of a bucco-antral fistula. Peter suggests a technic which, he says, can avert the development of such a fistula. It is considered that an osseus surface sufficient for supporting the severed soft tissues is necessary for proper adhesion and healing by first intention. Therefore the alveolar process is used as a zone complementary to the supporting surface. To this end an incision through the free margin of the gingiva is made for exposure of the antrum of Highmore. While the interdental papillae are readily damaged by a blunt trauma, severing with an edged instrument does not hinder excellent healing. The disadvantage of the method lies in the fact that the flap from the gingival margin is larger than that resulting from the usual incision, which makes the antrum of Highmore less accessible; besides, it may be difficult to avert an injury to the gingival margin while the latter is fixed with a surgical hook. These disadvantages are overcome by experience. Penetration of the instrument into an alveolus, between the tooth and the alveolar wall, and an exaggerated gingival flap are errors committed by the inexperienced; these errors are responsible for the criticism of the technic suggested. The direction of the suture line with regard to the wound is considered as another important factor in healing by first intention. Therefore the mucosal incision and the suture ought to be situated lateral to the opening of the wound; then rupture of the suture or infection of the channel produced by the puncture will not be a menace to the wound. The details of the technic and fifteen illustrations are contained in the article.

EDITOR'S ABSTRACT.

Society Transactions

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY

Nov. 19, 1930

H. P. Schenck, M.D., *Reporter*

George M. Coates, M.D., *Chairman*

Presentation of a New Type Tracheotomy Tube with Valve. Dr. Gabriel Tucker.

Most tubes devised for the treatment for laryngeal stenosis possess arrangements of the valves which occupy too much space. The ideal tube for emergency tracheotomy is the Jackson tube, because it is sufficiently long to reach the trachea. After the wound has healed, a different proposition is faced; the Jackson cannula now touches the posterior wall, as shown in roentgenograms. The Luer type of cannula is found to strike the anterior wall of the trachea. The new tube presented is intermediate in length between the Jackson and the Luer tubes, is fitted with a valve that functions in a number 4 tube and serves its purpose better than the older types now in use.

The Present Status of the Intracarotid Method of Treatment for Pneumococcic and Streptococcic Meningitis. Dr. John A. Kolmer.

Pneumococcic meningitis and streptococcic meningitis are still rare in diseases of the ear and the sinuses, yet the mortality is so high that this factor is impressive. Experimental work on dogs and cats demonstrated that by "quadruple treatment" survival of 30 per cent of the infected animals was possible. By continued efforts, an intracarotid method of injecting serum and drugs into the common carotid arteries was developed on the assumption that such a method would bring therapeutic agents into immediate contact with the meninges. From 40 to 60 per cent of the animals with experimentally produced meningitis recovered with the new treatment.

In man, the technic was employed in seven cases of hemolytic streptococcic meningitis and in four cases of pneumococcic meningitis. Not much was accomplished with the streptococcic infection because of the lack of a suitable antistreptococcic serum. One patient with pneumococcic meningitis, secondary to ethmoiditis, recovered. In this case the pneumococcus proved to be type I. A small child and two adults with well advanced meningitis, cloudy spinal fluid and several thousand cells per cubic centimeter in the spinal fluid died. Of these, one was moribund when the intracarotid injection was started. In the successful case, the patient was given the antistreptococcus serum of Felton, which contains a large amount of type I, a small amount of type II, a trace of type III and no type IV antibodies. Felton's serum is dispensed in syringes containing from 10 to 12 cc. (at least 10,000 units of antibody for type I pneumococcus).

The serum is injected into each common carotid artery, exposed surgically, a no. 22 needle and a syringe being used. There is practically no hemorrhage, and no ligature is required. From 10 to 12 cc. of serum is injected into each carotid, and merely a guide ligature is left under each artery; guide clamps are left in place, because two or three injections may be needed daily. One cubic centimeter of a 1:200 solution of ethyl hydrocuprein hydrochloride may be added to each syringe.

Four cases with one survival is a disappointingly low percentage, but if the technic could be applied as early to human beings as to experimental animals, the results should be equally good. Hesitancy to resort to such radical methods and the resultant delay are fatal. Emphasis must be laid on early diagnosis. As soon as pneumococci are found in the spinal fluid, the more radical plan of treatment should be tried immediately, regardless of any other observation, otherwise the patient will die anyway. Ordinary intraspinal treatment is a failure.

During the last year, streptococcic meningitis has been more common than pneumococcic meningitis. Reliable scarlet fever antitoxin is useful in treating patients with streptococcic meningitis, and is better than the polyvalent serums. Most infections from the mastoid are hemolytic and extremely toxic. Ordinary polyvalent serums, Pregl's solution, gentian violet, metaphen and neutral acriflavine, are as safely given by injection into the intracarotid arteries as by intravenous or intraspinal injection. In addition, there is an immediate contact with the meninges. After the introduction of from 10 to 12 cc. of scarlet fever antitoxin into each carotid, there is an almost immediate improvement. The spinal fluid shows a sharp reduction of pus cells per cubic centimeter, and on three occasions it was thought that recovery would occur. One patient died of abscess of the frontal lobe later. Further developments must await the preparation of more potent serums. As far as chemotherapy is concerned, only the surface is being touched and more powerful agents are needed. Intracarotic administration marks a distinct advance in the treatment for meningitis. The method is reported to have saved the lives of several patients with meningococcic meningitis resistant to intraspinal medication.

THE SPECIFIC PROPHYLAXIS OF PNEUMOCOCCIC AND STREPTOCOCCIC MENINGITIS. DR. JOHN A. KOLMER and DR. K. W. AMANO (by invitation).

It was found that when pneumococci or streptococci were injected into the cisterna magna of animals, they gave evidence of meningitis in from ten to fifteen hours and died in two or three days. Cultures introduced into the tympanic cavities of monkeys produced otitis media, but in no instance meningitis. When type I, Felton's pneumococcic serum (0.5 cc. per kilogram of body weight) was also administered, the onset of meningitis was delayed and death did not occur for five days and fifteen hours. The control animal died in twenty hours. It was found that 0.2 cc. of serum with 30 mg. of ethyl hydrocuprein hydrochloride per kilogram of body weight, when administered at the time of the infection, enabled the animals to survive for three or four days, while the controls died in from thirteen hours to one day and twelve hours.

Most of the experiments were performed on rabbits because the introduction of cultures into the tympanic cavities was usually followed by meningitis. Untreated controls died in from two to five days. When 0.2 cc. of serum per kilogram was given intravenously, the majority survived indefinitely. The serum was given immediately after the streptococci had been placed in the tympanic cavity. When the serum was given intramuscularly at the same time the cavity was infected, one animal died in two days and three others survived. Invariably, the injection of 0.4 cc. of serum per kilogram of body weight enabled the animals to survive.

It seems possible that in man with type I pneumococcic infections derived from the ethmoidal or sphenoidal sinuses or the mastoid area, the early administration of type I serum might prevent involvement of the meninges. When ethyl hydrocuprein hydrochloride (30 mg.) and serum (0.2 cc.) were given intramuscularly, all of the animals survived. There was a complete series of survivals when the medication was given by way of the vein. It was evident that in type I pneumococcic infections, Felton's serum in doses of from 0.2 to 1 cc. per kilogram of body weight would offset meningitis in animals when given intramuscularly or intravenously. Whether the addition of ethyl hydrocuprein hydrochloride to the serum is of advantage is not clear. When given by mouth, some prophylactic action is noted, but the serum is the more effective agent of the two. Ethyl hydrocuprein hydrochloride is not effective alone.

Of the untreated controls with type II pneumococcic infections, 83 per cent died (average of one day and six hours). A similar group treated with Felton's serum had a mortality of 34 per cent. This would indicate that the treatment for type II pneumococcic infection requires at least twice the dosage used in type I infections. There is so little antibody against type III pneumococcus that 63 per cent of the rabbits treated died, while 66 per cent of the rabbits not treated died.

It is difficult to produce streptococcic meningitis in dogs and rabbits. The experiments on prophylaxis required the introduction of cultures into the cisterna magna. Untreated animals died in an average of from thirteen hours to one day, while those receiving scarlet fever antitoxin lived from two days and thirteen hours to five days, and three hours. From the standpoint of recovery, the percentage of success is not as impressive as in pneumococcus infections.

Streptococcic meningitis remains more lethal than the pneumococcic form because therapeusis is less effective. Bacteriologic studies must be made early. If pneumococci are found and meningitis is suspected clinically, it is recommended that Felton's concentrated antipneumococcus serum be given intravenously (10 cc. for an adult; 5 cc. for a child). Later the same dose may be given intramuscularly, the dose being repeated on the third day. Enough antibody may gain access to the meninges to prevent meningitis. Immediate bacteriologic examinations are essential, so that if pneumococcus type I or II is found something worth while may be done to prevent meningitis. If hemolytic streptococci are found, one of the scarlet fever antitoxins should be used. The latter may produce reactions, while the pneumococcus serums produce no reaction.

DISCUSSION

DR. GEORGE B. WOOD: I have not seen many cases of meningitis, but we all appreciate the work Dr. Kolmer is doing to help us combat these disastrous infections. The prophylactic point of view is instructive. We cannot give serum in every case of streptococcic otitis media, but when the pneumococcus is present, the matter is different. A patient with pneumococcic otitis media, with profuse discharge and a positive culture reported in twelve hours, was given Huntoon's antibodies; the pneumococci disappeared from the ear in two days. Huntoon's antibodies do not cause so severe a reaction that they cannot be given in cases of otitis media, and I believe that such measures should be used in treating patients with otitis media.

DR. AMANO: The tympanic cavity of the dog is so deeply placed that a surgical approach is necessary to infect it. In some instances the tympanic membrane may be curetted. In one series, following tympanic application, the untreated animals showed a mortality of 77 per cent, while the treated animals had a mortality of 13 per cent.

DR. FRANCIS C. GRANT: It is a pity that we do not have more researches of this type, well thought out at the beginning and carefully controlled. I wish to ask if some of my impressions are correct. In combating infection, the protective mechanisms of the body produce adhesions of the meninges, and in this event serum cannot be brought to the blocked off area by the intraspinal route. The only route of approach is the intracarotid. I have seen many infections of the mastoid that looked as if they might be meningitis for twenty-four hours, and then all blew over and nothing developed. We should use these methods early, but since we often see children with flare-ups that later prove to be meningismus and not meningitis, there is some hesitancy in using serum. I have never seen a patient with pneumococcic meningitis who recovered. We usually hope that the symptoms reflect a serous meningitis or meningismus and that therapy may not be required. This therapy, to be effective, must be used early. I have seen patients with pneumococcic meningitis do well for a time and then suffer from a relapse. Some infections of the mastoid progress to meningitis because the area of the mastoid has not been thoroughly cleaned. In such cases the

primary meningitis might be controlled by Dr. Kolmer's technic, and a second flare-up might result from the incompletely cleaned area of the mastoid and progress to meningitis. Sperling reported eight cases, with five recoveries due to laminectomy and permanent drainage through the laminectomy wound, with the administration of large quantities of water. We tried the same treatment, but all of our patients died. I should like to ask Dr. Kolmer if it is possible to infect the semicircular canals rather than the tympanic cavity.

DR. AMANO: Labyrinthitis is one of the first steps to meningeal complications. We do not know whether we caused labyrinthitis first, or if hematogenous spread occurred. In some of our animals injections were made into the labyrinth. We opened the cochlea of the dogs. In rabbits, pneumonia followed such procedures, so that we had to pass a needle through the overlying muscular structures and so penetrate the middle ear. The simplest method is administration through the drum. I am sure that labyrinthitis developed in many of the animals.

DR. E. B. GLEASON: A child may show symptoms of meningitis, and these symptoms may disappear after operation on the mastoid. When meningitis appears after an operation on the mastoid, it is likely that the meningitis will persist and progress.

DR. AMANO: The frequency of meningeal infections secondary to infection in the area of the mastoid is supposed to be about 1 in 300 or 1 in 150.

DR. GEORGE M. COATES: In answering Dr. Grant, I should like to say that we do not see many cases of meningitis after operation on the mastoid. There may be a cloudy spinal fluid, with 2,000 cells per cubic millimeter but no organisms, but the case clears up after operation and we assume that it was one of serous meningitis. Some cases do develop, however, and we cannot tell whether profuse suppuration will occur or whether the process will subside. In a study of 650 cases of infection of the mastoid, a mortality of from 3.5 to 4 per cent was found, and since many of these were late cases, there apparently were not many of meningitis. How many of us leave infection in the mastoid? We clean the mastoids much more thoroughly than we did twenty years ago, but, like dealing with ethmoidal cells, it is frequently hard to clear away every cell.

DR. RALPH BUTLER: Most cases of meningitis occur before operation on the mastoid. Dr. Kolmer has given us a good idea as to prophylactic measures. If we could use these methods early in cases of otitis media, they should prove effectual. Several patients with meningitis that occurred before operation were given intracarotid injections several years ago, but all of them died. Our serums may be more potent now.

DR. JOHN A. KOLMER: While the otolaryngologist is now able to leave a clean field after operation on the mastoid, it is much more difficult to remove thoroughly all of the infected cells in ethmoidal infections. I have seen many cases of ethmoidal infection in which the surgeon had to be "jacked up" about residual infection. Dr. Gleason emphasized the difficulty of diagnosis, and this is a point well taken. I know of at least two patients with pneumococcus meningitis who recovered, but the spinal fluid was clear in both instances and the pneumococci were gotten only in culture. This is not the type of case I mean. I mean that in which there is cloudy spinal fluid, with abundant organisms in the fluid. What are we going to do with a strongly positive globulin reaction and 1,000 cells per cubic millimeter in the spinal fluid? Shall we wait or use this therapy? I think that it is safer to operate and to use intracarotid therapy, because there is no danger to the patient. All surgeons doing work in otolaryngology are capable of using the carotid for this purpose.

TREATMENT FOR STRICTURE OF THE ORAL PHARYNX. DR. GEORGE M. DORRANCE.

Stricture of the oral pharynx results from the swallowing of acids or caustics; it occasionally occurs as a complication of ulcerative syphilis of the pharynx,

and, on rare occasions, it follows tonsillectomy. Congenital strictures at this location have not been reported. Treatment by gradual or rapid dilatation of the opening is unsatisfactory, as the scar tissue invariably contracts again.

The patient presented had a stricture of the oral pharynx following tonsillectomy. The opening in the pharynx was situated on the posterior surface of the tongue, and the edges of the stricture were rigid. Tracheotomy was done, and the opening permitted to close. An incision was made on either side of the stricture, and a cavity was made with a hemostat through the lateral wounds. The cavities were enlarged upward, and specially constructed vulcanite pegs were introduced, being first pushed well below the stricture and then pressed upward into the preformed cavity to prevent each peg from jumping out. The vulcanite capsules were then removed and washed. Two large Thiersch skin grafts were then cut and placed over the vulcanite capsules with the surfaces of the skin toward the capsule. The capsules were then reintroduced into the cavities and the wounds closed by sutures. The capsules were left in place for two weeks, when an incision was made on either side of the stricture down to and through the skin grafts and the capsules removed. A skin-lined cavity remained on either side of the previously existing stricture. By pressure, these cavities were converted into a single cavity. A small rubber ring pessary was then introduced and allowed to remain for two weeks. The pessary was removed for purposes of cleansing every few days. At the time of presentation, the patient was able to swallow a normal, mixed diet, and there was no respiratory difficulty, approximately two and one-half years after the operation.

DISCUSSION

DR. FIELDING O. LEWIS: I have nothing to add. The patient received all sorts of treatment. Electrocoagulation and fulguration were tried without success. I can vouch for all Dr. Dorrance has said about the recovery.

DR. GEORGE M. COATES: Does the presence of the skin grafts affect swallowing? How does skin act in the moist medium of this region?

DR. GEORGE M. DORRANCE: The tissue resembles mucous membrane now, and under the microscope looks like neither mucous membrane nor skin. It is moist, just like mucous membrane. I have not been able to try this technic in cases of caustic contracture.

Dec. 17, 1930

H. P. SCHENCK, M.D., *Reporter*

GEORGE M. COATES, M.D., *Chairman*

ORBITAL ABSCESS FOLLOWING DISEASE OF THE SINUS. DR. JOHN R. DAVIES, JR.

A girl, aged 14 years, had had her tonsils and adenoid tissue removed at the age of 2 years. She was not especially subject to colds in the head, but when they occurred they tended to be prolonged. When first examined, the girl had had a cold in the head for ten days, and dating from the preceding day there had been considerable edema of the right eyelid, some protrusion of the eyeball and a slight edema of the conjunctiva. The patient was decidedly toxic and had much pain over the right antrum. The mucosa in the right nostril was swollen, and the nostril was filled with pus. The temperature was 101 F.

The roentgenologic report from wet films indicated that there was pus in the right antrum, but the picture of the right frontal and ethmoidal sinuses was indefinite. The leukocyte count was 6,200, and the urine was loaded with albumin and erythrocytes. Because of the age and sex of the patient and of the vague

roentgenologic picture the frontal sinus was not opened. A Caldwell-Luc operation revealed considerable pus and many polypi in the antrum. *Staphylococcus aureus-hemolyticus* was recovered from the pus in pure culture. The following morning there was increased swelling of the right eyelid, marked edema of the conjunctiva and protuberance of the eyeball. The right frontal sinus was opened through the usual incision in the eyebrow, and considerable pus was evacuated. The roof of the orbit was opened, and free pus found. Little pus was found in the ethmoidal cells.

The incision in the eyebrow was closed with dermal sutures, and drainage was maintained intranasally. Twenty-seven days after admission to the hospital, when the patient was discharged, the ocular movements and vision of the right eye were normal. A year after operation, the sinuses were clear on transillumination, and the eye was normal.

The case illustrates difficulties frequently met in the early diagnosis of sinus disease, in the absence of headache or pain over the sinus on pressure. With a history of repeated or prolonged colds in the head, with vague roentgenologic reports and an indefinite picture in the nose and on transillumination, is the exploratory puncture of the antrum a safe and logical procedure, and might it have prevented this complication?

DISCUSSION

DR. J. THORINGTON: This case was the most remarkable of its type that I have ever encountered. The conjunctiva was protruding between the lids, the retinal vessels were tortuous and full, but fortunately no break had occurred in the vessels. It was interesting to observe how rapidly the edema disappeared. There had been proptosis of a half inch on the affected side and some on the opposite side.

DR. FIELDING O. LEWIS: In this connection I should like to mention the case of an infant 15 days old with proptosis due to disease of the ethmoidal sinuses. The diagnosis was corroborated by roentgenologic and operative observations, but the patient died.

DR. GEORGE FETTEROLF: A somewhat similar case was seen at the University Hospital. There was some edema of the tissues about the eye on the uninvolved side, and at operation an opening was found in the interfrontal septum. I was curious as to whether such an opening in the interfrontal septum was found in this case at operation.

DR. JOHN R. DAVIES: The interfrontal septum was found to be intact at operation.

ACUTE MASTOIDITIS WITH BASILAR MENINGITIS. DR. WILLIAM HEWSON.

A man, aged 41, developed acute otitis media on the right side about nine weeks after an acute cold. Incision was promptly made, and after draining for four weeks, a furuncle developed in the right external auditory canal, followed by a postauricular abscess. Drainage of these abscesses continued for two weeks accompanied by a persistent headache on the right side and intermittent nausea. There was no vertigo. At this time the patient was admitted to the hospital, the temperature was 100.6 F., the pulse rate 94 and respiratory rate 22. The pupils reacted to light and accommodation, and there was no impairment of ocular movements. There was a little tenderness over the area of the mastoid, and the external canal was a trifle edematous. The drum membrane was inflamed and slightly bulging and was reincised. The nasal septum was deflected in its midportion, crowding the middle turbinate on the right. Mucopus was visible in the middle and inferior meati on the right. The pharynx was congested as well as a small residual tab of the right tonsil.

A roentgenogram revealed uneven cloudiness throughout the area of the right mastoid and localized areas of absorption in the antrum in the region of the knee of the sinus toward the tip, the appearance suggesting acute suppurative mastoiditis. From the second to the eighth day in the hospital there was improve-

ment. On the eighth day nausea and vomiting appeared, with a chill and temperature of 102 F. The following day there was stiffness of the neck and increased reflex response. The leukocytes were 24,000, with 95 per cent polymorphonuclears. The spinal fluid, under a pressure of 40 mm., was cloudy and contained 12,000 leukocytes.

At operation, the cortex of the mastoid was firm and thick. A fair-sized abscess cavity occupied the tip of the mastoid, but the lateral sinus was found to be normal. In following the zygomatic cells, a necrosed area was reached, which communicated by a fistulous tract throughout the aditus ad antrum. The dura over this area was inflamed and adherent, and a small perforation was found in the dura opposite the fistulous tract. The wound was left open and packed. The following day the temperature rose to 105 F., and the patient died. Pneumococci were cultured from the spinal fluid, and at autopsy a localized pachymeningitis was found to involve the pons and medulla, especially on the right. An area of necrosis involved the petrous portion of the temporal bone.

This case illustrates the possibility of an acute rhinitis developing into an ethmoiditis, with extension up the right eustachian tube to the middle ear to produce purulent otitis media. Cells in the immediate vicinity of the aditus were probably first attacked, and extension involved the adjacent cells in the petrous pyramid. The breaking down of cells in the posterior root of the zygoma made the fistulous tract which extended through the periosteum at this point. Extension is thus possible from mastoid cells to the cells in the petrous pyramid.

DISCUSSION

DR. LOUIS BAER: I recently had a case similar to the one reported by Dr. Hewson. I should like to know when unconsciousness came on. My case was due to the streptococcus. The drain was taken from the wound on the ninth day. On the eleventh day, there was headache and a temperature of 99 F. The drain was reinserted and taken out two days later. For five days there was a temperature of about 99 or 99.5 F. At consultation it was thought there might be some retention. The following morning the patient could not be aroused. The streptococcic type is more virulent than the pneumococcic type, and there is less likely to be recovery. Serums, vaccine, bacteriophage and other measures should be used early to prevent extension of the infection.

DR. B. ALEXANDER RANDALL: Pachymeningitis is a mild condition, and with drainage recovery usually results. It is only with penetration of the arachnoid structures, with arachnoid involvement, that there is danger. In pachymeningitis there is thickening of the membrane and granulations form on the outer surface which are more impervious to infection than the usual membrane. Frequently actual penetration and opening up of the arachnoid cavity occur, as in this case with pus in the spinal fluid, and only vigorous treatment will avail. I believe much can be done by washing out these cavities, even using a spatula which will push the brain tissue aside. I have washed out large quantities of thick pus in this fashion. It did not always save the patient, but much may be accomplished in this way.

DR. FIELDING O. LEWIS: I should like to ask Dr. Hewson if the cultures were from the mastoid or the cerebrospinal fluid.

DR. HEWSON: The culture was from the cerebrospinal fluid, but the same organism was obtained from the mastoid. The patient was conscious almost until death. Dr. Shuster saw this case with me, and I should be glad to hear from him.

DR. B. H. SHUSTER: The first sign of meningitis was the development of chill. Nausea preceded the other signs of severe illness. Undoubtedly this patient had pachymeningitis early, but at that time no other symptoms of meningitis could be elicited. The furuncle did not clear up soon after incision, leading to the suspicion that necrosis of the bone might be responsible for its persistence. Many patients with early symptoms similar to those shown by this man recovered.

DR. FIELDING O. LEWIS: It occurred to me that this would have been a suitable case in which to employ the Kolmer treatment.

COMPOUND FRACTURE OF THE MALAR BONE WITH INVOLVEMENT OF THE MAXILLARY SINUS. DR. THOMAS G. AIKEN.

The patient who was present, had received a violent blow over the right malar bone, resulting in a compound fracture which later became infected. For six weeks the temperature remained elevated and then fell but never entirely reached normal. Dr. Aiken first saw the patient at this time, and a roentgenogram revealed the extent of the fracture and indicated increased cloudiness of the antrum. A needle introduced into the antrum released pus, and after a series of irrigations the temperature returned to normal. Later the patient returned with a swollen face and toothache. A dentist extracted one molar tooth having two normal roots and a third root surrounded by a large granuloma. No direct communication of the socket of the tooth with the antrum could be demonstrated. Two weeks after the extraction, the pus had almost disappeared, and at the time of presentation there was no pus in the antrum; on transillumination the sinus was found to be much clearer.

DISCUSSION

DR. GEORGE B. WOOD: We may expect almost anything from traumatic infection of the sinuses. One patient who was admitted to the Graduate Hospital, in a state of unconsciousness had been struck three times with a hammer. One blow had struck the frontal area, one the maxillary sinus and the third the area of the mastoid. This area was wide open; the frontal sinus was open, and the eye had dropped out so that the front walls of the sphenoidal sinuses could be seen through the smashed walls of the ethmoidal cells. When the wound was dressed a tremendous cerebrospinal leak was found. Dr. Grant made an osteoplastic flap and lifted the brain from the cribriform plate after turning the dura back from the frontal lobe. The leak could not be found so the wound was closed, and the woman later made a complete recovery.

DR. FIELDING O. LEWIS: I saw a woman with a fractured outer wall of the maxillary sinus due to an automobile accident. A very foul and extensive infection followed which later required a radical operation for relief.

CHONDROMATA OF THE LARYNX: REPORT OF A CASE. DR. ROBERT M. LUKENS.

Seventy-one cases of chondromas of the larynx are reported in the literature. They most frequently appear in the cricoid or the thyroid cartilage and usually are sessile and single. They show a tendency to invade adjacent cartilages, but rarely, if ever, metastasize. Masses of new tissue appear, composed of hyaline, elastic or white fibrocartilage, developing from preexisting cartilage. The tumor is usually rounded, smooth, tuberous or lobulated with well marked capsule. According to many, occurrence is rare after puberty, but the cases reported occurred in persons from 15 to 66 years of age so that chondromas of the larynx are tumors of rather late adult life. Even with complete shelling out of the tumor, recurrence is apt to occur, and laryngectomy is required more often than is supposed.

A white man, aged 57, was rejected by an industrial medical examiner because of husky voice and the presence of a tumorlike mass in the right side of the neck. The hoarseness was of two years' duration, but there had been no cough, pain, dyspnea or dysphagia. To the right of the midline of the neck at the level of and above the cricoid cartilage, was a fairly smooth, hard, bonelike, hemispherical mass about seven-eighths inch in diameter and about one-half inch thick, firmly attached to the anterior arch of the cricoid and lower portion of the right thyroid ala. Mirror laryngoscopy revealed a marked prominence of the subglottic tissues on the right side. There was no noticeable impairment of motion of the overlying cord except lack of tension of both cords on phonation. No ulcerations were present.

The tumor mass in the neck was removed by external operation under local anesthesia. The patient made a good recovery, but some time later attacks of dyspnea with inspiratory crowing sounds developed. Three years after the removal of the tumor, the laryngeal lumen was greatly narrowed by a rounded mass, which projected inward from the right side, obliterating the anterior portion of the chink, raising up the right vocal cord and pushing it inward so that the right ventricle could be inspected readily. The arytenoid was not involved. The subglottic tissues on the left were not involved.

Laryngectomy was indicated but postponed because of a severe infection of the mouth. Tracheotomy was necessary because of the extreme dyspnea. Later, laryngectomy was performed and the tissue found to be that of a chondroma. A good recovery was made, but the patient became unmanageable later, and died of bronchopneumonia eight days after the operation.

Book Reviews

TUMORS DEL NASOFARINX. By ANTONIO E. GAY, M.D., Pp. 74. Buenos Aires, La Semana médica, 1930.

This volume of 74 pages is a doctoral thesis. In the first three chapters, anatomic, physiologic and histologic considerations of the nasopharynx are presented. There is also a chapter concerning anatomicopathologic studies on the several types of tumors that may develop in the nasopharynx. The methods in use for the examination of the nasopharynx as well as for the differential diagnosis are reviewed. The author believes that the histologic constitution of the nasopharynx is the substratum for the several anatomicopathologic forms of nasopharyngeal tumors the development of which is favored by unknown causes. The architecture, physiology and anatomic relations of the nasopharynx explain the symptoms presented by tumors that develop in that region. The symptoms given by nasopharyngeal tumors point out the presence of any of the following five clinical forms of tumors: the nasal, the auricular, the ganglionar, the ocular and nervous and the mixed form. The mixed form is a combination of two or more of the four clinical forms. This form is observed during the period of evolution of tumors of the nasopharynx, and its symptomatology depends on the type of tumor. The importance of early diagnosis and treatment is considered in relation to the prognosis. Prognosis, as well as treatment, depends on the benign or malignant character of the tumor. Special reference is made to diathermic coagulation, either by itself or as a combined treatment with radium therapy and deep roentgen therapy for malignant tumors. The clinical histories of thirteen patients, who were observed by the author, are also given. The book contains many illustrations, including operative instrumentation, histologic reproductions and pictures of patients. It will be read with interest. The bibliographic notes however, point out that the author focused his attention especially on Argentina literature, though some French literature is also included.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. T. E. Carmody, 227, 16th St., Denver.
Secretary: Dr. Gabriel Tucker, Bronchoscopic Clinic, University Hospital, Philadelphia.
Place: Philadelphia. Time: June 8-12, 1931.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. William H. Wilder, 122 S. Michigan Ave., Chicago.
President-Elect: Dr. J. F. Barnhill, Miami Beach, Fla.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: French Lick, Indiana. Time: Sept. 14-19, 1931.

American Bronchoscopic Society

President: Dr. Henry B. Orton, 24 Commerce St., Newark, N. J.
Secretary-Treasurer: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia.
Place: St. Louis. Time: June 6, 1931.

American Laryngological Association

President: Dr. Francis R. Packard, 304 S. 19th St., Philadelphia.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Place: Atlantic City, N. J. Time: June 15-17, 1931.

American Laryngological, Rhinological and Otological Society

President: Dr. Max A. Goldstein, 3858 Westminster Pl., St. Louis.
Secretary: Dr. R. L. Loughran, 33 E. 63d St., New York.

American Otological Society

President: Dr. David H. Walker, 101 Bay State Rd., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.

SECTIONAL

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. G. G. Marshall, 122 West St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

New England Otological and Laryngological Society

President: Dr. Harry P. Cahill, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Lyman G. Richards, 319 Longwood Ave., Boston.

* Secretaries of societies are requested to furnished the information necessary to make this list complete and to keep it up to date.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Chester H. Bowers, 1136 W. 6th St., Los Angeles.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Albert E. Hillis, 1115 Pacific Ave., Tacoma, Wash.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Fletcher D. Woodward, Box 162, University, Va.
Secretary: Dr. W. D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: New Orleans. Time: November, 1931.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. Wilfrid Haughey, 65 W. Michigan Ave., Battle Creek.
Secretary: Dr. R. H. Fraser, Battle Creek Sanitarium, Battle Creek.
Time: Third Thursday of alternate months.

STATE

COLORADO OTO-LARYNGOLOGICAL SOCIETY

President: Dr. C. E. Cooper, 227, 16th St., Denver.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Dorland Smith, 836 Myrtle Ave., Bridgeport.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. William L. McDougall, 50 Whitehall St., S. W., Atlanta.
Secretary-Treasurer: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. John C. Brown, 350 St. Peter St., St. Paul.
Secretary-Treasurer: Dr. Hendrie W. Grant, Lowry Bldg., St. Paul.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. Lee W. Smith, 125 W. Granite St., Butte.
Secretary: Dr. J. G. Parsons, Lewistown.
Time: Semi-annually.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. H. B. Beeson, 232 W. De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. W. McCollom, 346½ Washington St., Portland, Ore.
Secretary: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland, Ore.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Raymond F. Hacking, 105 Waterman St., Providence.
Secretary: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Erza Thompson Bldg., Salt Lake City.

VIRGINIA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Fletcher D. Woodward, Box 162, University.
Secretary-Treasurer: Dr. Henry Grant Preston, Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. S. S. Hall, Fairmont.
Secretary: Dr. G. A. Smith, Montgomery.
Place: Clarksburg. Time: May 19-21, 1931.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. J. Wallace Hurff, 86 Washington St., Newark.
Secretary: Dr. Earl LeRoy Wood, 31 Lincoln Park, Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. John R. Childs, 45 Edgewood Ave., Atlanta.
Secretary: Dr. William O. Martin, Jr., Medical Arts Bldg., Atlanta.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE CITY MEDICAL SOCIETY, SECTION ON OTOLOGY AND LARYNGOLOGY

Chairman: Dr. William R. McKenzie, 11 E. Chase St., Baltimore.
Secretary: Dr. James J. Chisolm, Medical Arts Bldg., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. George J. Dennis, 25 E. Washnigton St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

Cincinnati Oto-Laryngological Society

President: Dr. Edward D. King, 707 Race St., Cincinnati.
President-Elect: Dr. M. F. McCarthy, Union Central Bldg., Cincinnati.
Secretary-Treasurer: Dr. Samuel Seltz, Provident Bank Bldg., Cincinnati.
Place: University Club. Time: 6:45 p. m., second Tuesday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. William V. Mullin, 9204 Euclid Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 2064 E. 9th St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

College of Physicians of Philadelphia, Section on Otolaryngology

Chairman: Dr. Ralph Butler, 2007 Chestnut St., Philadelphia.
Clerk: Dr. Karl M. Houser, 1826 Pine St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

Columbus Ophthalmological and Otolaryngological Society

Chairman: Dr. A. M. Hauer, 327 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Albert D. Frost, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Otolaryngology

President: Dr. H. B. Decherd, 1717 Pacific Ave., Dallas, Texas.
Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Detroit Oto-Laryngological Society

President: Dr. Don A. Cohoe, 13535 Woodward Ave., Detroit.
Secretary-Treasurer: Dr. R. Lee Laird, 513 David Whitney Bldg., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. J. J. Richardson, 606 Penn St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Claude C. Cody, 1306 Walker Ave., Houston, Texas.
Secretary: Dr. J. M. Robinson, Eye, Ear, Nose and Throat Hospital, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. E. L. Lingeman, 241 N. Pennsylvania St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. A. E. Eubank, Arygle Bldg., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Dean E. Godwin, Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Ferris Arnold, Security Bldg., Long Beach, Calif.
Place: Pacific Coast Club. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section of Ophthalmology and Otolaryngology

President: Dr. Eugene R. Lewis, 727 W. 7th St., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. 7th St., Los Angeles.
Place: California Hospital, assembly room. Time: 8 p. m., first Monday of of each month, May to November, inclusive.

Medical Society of the County of Kings, Section on Otolaryngology

Chairman: Dr. John M. Auwerda, 878 President St., Brooklyn.
Secretary-Treasurer: Dr. Charles R. Weeth, 37 Remsen St., Brooklyn.
Place: Library Bldg., County Society, 1313 Bedford St., Brooklyn.
Time: 9 p. m., second Wednesday of January, March, May and November.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. James A. Flynn, 1511 Rhode Island Ave., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St., N. W.: Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

The Milwaukee Oto-Ophthalmic Society

President: Edward R. Ryan, 425 E. Water St., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 120 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville.
Secretary-Treasurer: H. C. Smith, Medical Arts Bldg., Nashville.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

New Orleans Ophthalmological and Otological Society

President: Dr. W. R. Buffington, 211 Camp St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: January to June and October to January—third Thursday of each month.

New York Academy of Medicine, Section of Laryngology and Rhinology

Chairman: Dr. John M. Loré, 240 West End Ave., New York.
Secretary: Dr. Robert E. Buckley, 2 E. 103d St., New York.
Place: 103d St., and 5th Ave. Time: 8:30 p. m., fourth Wednesday of each month from October to May.

New York Academy of Medicine, Section of Otology

Chairman: Dr. Clarence H. Smith, 2 E. 54th St., New York.
Secretary: Dr. James Garfield Dwyer, 57 W. 57th St., New York.
Place: 103d St. and 5th Ave. Time: Second Friday of each month from October to May.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. Charles M. Swab, Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. J. C. Davis, 1615 Howard St., Omaha.
Place: Medical Arts Tea Room. Time: 5:15 and 7 p. m., third Wednesday of each month from October to May.

Philadelphia Laryngological Society

President: Dr. Arthur J. Wagers, 1429 Spruce St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

Pittsburgh Otological Society

Chairman: Dr. Watson Marshall, 121 University Pl., Pittsburgh.
Secretary-Treasurer: Dr. Thomas B. McCollough, 121 University Pl., Pittsburgh.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. Clyde Heatly, 11 N. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. John F. Gipner, 53 S. Fitzhugh St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ear, Nose and Throat Club

Acting President: Senior member of the executive committee: Dr. George E. Hourn, 607 N. Grand Blvd., St. Louis.
Secretary: Dr. Arthur M. Alden, Frisco Bldg., St. Louis.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. T. J. Walthall, 728 Main St., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Wilber F. Swett, 693 Sutter St., San Francisco.
Secretary: Dr. Robert Carson Martin, 384 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Third Tuesday of every month except May, June, July and December.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. Paul A. Remington, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. Searle B. Marlow, State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubinstein, Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

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